THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—41ST YEAR SYDNEY, SATURDAY, JANUARY 9, 1954 No. 2

Table of Contents.

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— Page.

The Stawell Oration—Tomorrow's Engineering Development in Victoria, by Major-General Sir Clive Steele, K.B.E., D.S.O., M.C., V.D. 25

Early Effects of Lead on Lymphoid Cells, by D. O. Shiels 30

The Association Between Hypoglycæmia and Myocardial Infarction, by Bryan Gandevia 33

Disseminated Neurodermatitis and Cataract, by R. Hertzberg, F.R.A.C.S. 36

REPORTS OF CASES—

A Report of Two Cases of Volvulus of the Small Intestine, by R. I. Mitchell 38

A Case of Probable Ovarian Agenesis with Turner's Syndrome in a Female Child Aged Seven Months, by Elizabeth K. Turner, M.D. 39

REVIEWS—

Incompatibility in Prescriptions 43

Dermatologic Formulary 43

The Spinal Cord 43

Trends of Life 43

Hormonal Factors in Carbohydrate Metabolism .. 43

Pathology of Tumours 43

William Cheselden 44

NOTES ON BOOKS, CURRENT JOURNALS AND NEW APPLIANCES—

Australasian Annals of Medicine 44

Family Doctor 44

LEADING ARTICLES—

Atherosclerosis 45

CURRENT COMMENT—

Effects of Smoking on Nutrition 46

Sudden Death during Infancy 47

Chairs and Sitting 47

CURRENT COMMENT—Continued. Page.

Chemotherapeutic Management of Infantile Diarrhœa and Vomiting 48

Lactation and Fluid Intake 49

ABSTRACTS FROM MEDICAL LITERATURE—

Therapeutics 50

Neurology and Psychiatry 51

SPECIAL ARTICLES FOR THE CLINICIAN—

XCII. Tonsillectomy 52

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific 54

OUT OF THE PAST 61

OBITUARY—

Edmund Britten Jones 61

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA 63

NAVAL, MILITARY AND AIR FORCE—

Appointments 63

POST-GRADUATE WORK—

The Post-Graduate Committee in Medicine in the University of Sydney 64

DEATHS 64

DIARY FOR THE MONTH 64

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 64

EDITORIAL NOTICES 64

The Stawell Oration.[1]

TOMORROW'S ENGINEERING DEVELOPMENT IN VICTORIA.

By MAJOR-GENERAL SIR CLIVE STEELE, K.B.E., D.S.O., M.C., V.D.,
Melbourne.

I REGARD it as a great privilege to have been asked to give this oration tonight.

The Royal Australian Army Medical Corps has given, to my personal knowledge, unrivalled service to the Army in two World Wars. In their skill, their thoroughness, and their unremitting devotion to duty, the members of your profession have been second to none. Whilst one must naturally attribute this partly to the personalities of those involved, one must recognize and appreciate that, basically, proper training has brought out all that is best in those personalities.

In this oration in memory of Sir Richard Stawell we do honour to a truly great teacher of medicine, one whose life work lives on for the good of the community, and whose traditions of service will always be an inspiration to those who follow him.

In speaking to you on the future of engineering in the State my object is to give, in not too technical a fashion, an outline of what my profession is doing and will do. The subject is a large one. I hope that I can do it justice and yet be merciful to my listeners.

Most of the big developments in Victoria are in control of government departments, and I have to thank those in charge of them for their courtesy in helping with much of the material necessary in preparing this paper.

In my opinion there should be no basic difference between government instrumentalities and big companies. (I am naturally referring to organization, not to taxation.) In either case Parliament, or the board, should (i) determine policy, (ii) ensure proper initial organization within the department or company, (iii) ensure good management, (iv) ensure continuity of management.

In all cases policy should be carried out by a chief executive officer, generally free from interference, but responsible to the higher authority for carrying out policy. It is the prime duty of governments and of boards of directors to ensure that their chief executive officer has authority, financial and otherwise, commensurate with his responsibility. Some government departments have such a proper charter; others unfortunately are subject to an intermediate control, such control having authority over the department but no responsibility for the work done.

[1] Delivered at a meeting of the Victorian Branch of the British Medical Association on October 7, 1953.

I should like also to mention administration. Most executive heads of our government departments are engineers. One occasionally reads statements to the effect that we should have administrators. Administration is basically common sense—such as the ability to select subordinates, to delegate authority to them, to be loyal to them, and yet to appreciate what goes on, and to have the *savoir-faire* to know that one can directly control only a limited number of subordinates. An engineer with that sort of common sense is obviously better in charge of a technical department than even the best administrator from a chain store.

sary in this area involving 1,000,000 acres—that is, one and a half times greater than the triangular area contained by Melbourne, Ballarat and Geelong.

Peculiarly enough, scientists originally discovered the soil deficiency in the desert by analysis of the wiry type of wool from sheep lost in the desert, not by analysis of the soil. But, once we have arrived at the scientific treatment necessary, the job is essentially one for earth-moving equipment.

I should like to interrupt myself at this stage by pointing out how much mechanization means to all industry. For

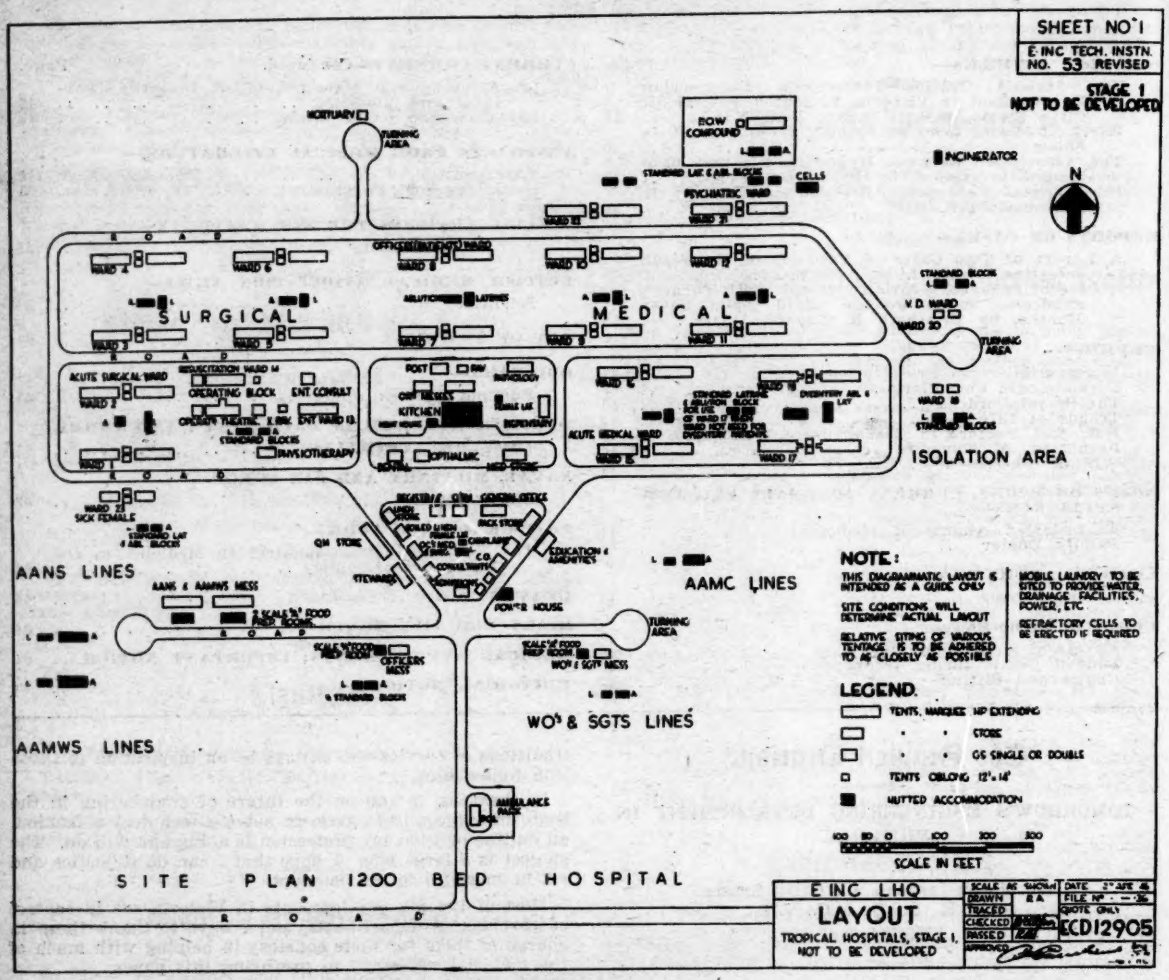


FIGURE I.

So far this has apparently had little to do with the future of the State; but a little thought will indicate that it has a great bearing on its orderly progress.

To turn now to the subject matter. Whilst Victoria must progress as a whole, its main immediate developments lie in the following: (i) land development, such as that of the desert north of Kaniva and west to Keith in South Australia; (ii) power and industrialization in the Latrobe Valley; (iii) irrigation in the Goulburn Valley; (iv) incidentals thereto—railways, roads, ports *et cetera*.

Land Development.

I will deal with the A.M.P. scheme (Kaniva–Keith) first. Primarily science has made much engineering work neces-

instance, one rated horse-power, where it can be applied, can do the work of two to three men. A two horse-power pump will pump as much water as five men; a 100 horse-power bulldozer will do the work of 250 men. To come closer home, a quarter horse-power motor will drive a drill and assist the dentist considerably, whereas previously he was largely occupied with working the treadle. This ratio of one horse-power to two and a half men was one of the main contributors to winning the war in the Pacific. A bulldozer—that is, a tractor with a blade in front—is much easier to feed and to hospitalize than the several hundred men it replaces.

Let us revert now to the Kaniva–Keith scheme. The land is treated in the following stages: (i) The ground is

cleared by tractors working in parallel and dragging either one or two big logs or a heavy chain about 400 feet long, in a rough semicircle between them. This flattens the scrub, and the work is done at the rate of 100 acres per hour. (ii) The uprooted scrub is allowed to dry off for six months and then burnt. (iii) The ground is then ploughed by a tractor towing two "Majestic" ploughs. These ploughs are very heavy, are peculiar to Australia, and are made in Adelaide. This work is done at the rate of 80 to 100 acres per day. (iv) The ploughed ground, which has deep furrows, is then smoothed by a tractor dragging a leveller weighing several tons and fabricated out of tram rails. (v) The ground is then given a treatment of one bag

. . . I have examined the use to which the good rainfall areas of each State are now being put . . . .

Certain outstanding conclusions can be derived . . . .

1. That even in the most highly developed State—Victoria—and allowing for a 20 per cent. deduction to cover land of no use in agriculture or for forestry, there still remains a total of 18,000,000 acres of unimproved lands . . . . This compares with 16½ million already developed.

2. In New South Wales there are 76 million such acres.

3. That in Queensland we have the staggering total of 190 million acres yet undeveloped.

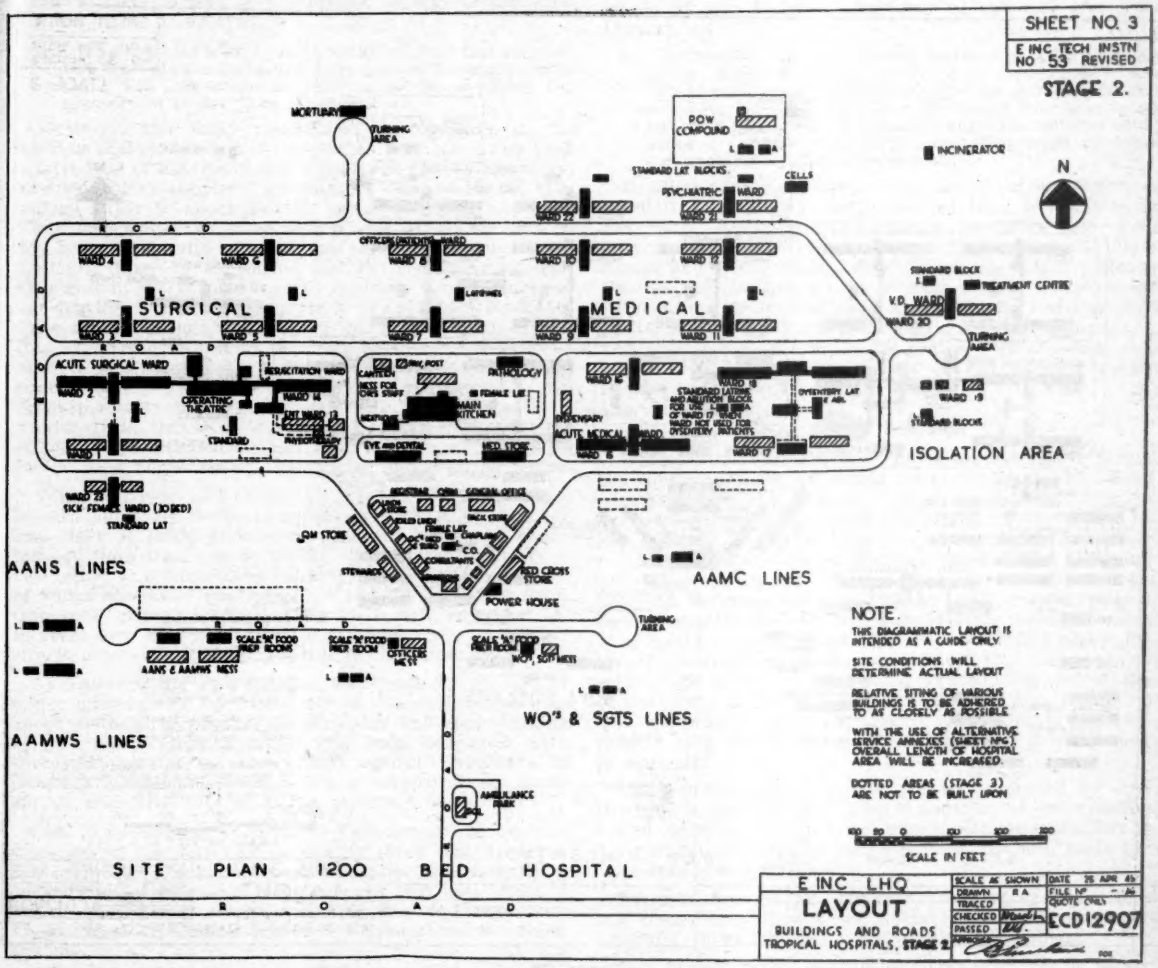


FIGURE II.

of superphosphate to the acre, about 7·5% being trace elements, in this case, zinc and copper sulphate.

The ground is then ready for planting with various types of grasses. Incidentally, Bacchus Marsh clover is one of the most successful. From desert, this country has become able to carry one and a half sheep to the acre.

The significance of trace elements to Victoria and Australia generally will be appreciated by the following statement, condensed from the presidential address of J. Griffiths Davies, Ph.D., of the Commonwealth Scientific and Industrial Research Organization, to the Australian Institute of Agricultural Science at Canberra in 1952:

Thus, briefly, one-third of the area of Victoria, and in New South Wales and Queensland approximately an area of five times the total area of Victoria, can be made productive merely by trace elements.

T. A. Lang, M.C.E., formerly engineer for design in the State Rivers and Water Supply Commission, estimated some years ago that the ultimate self-supporting population of Australia was 25,000,000 people. This was based on water conservation. Trace elements have therefore increased this figure very considerably. It may be of interest to know that if Victoria was settled at the same density rate

as the arid country of Israel, which many of us know, its population would be of the order of 20,000,000.

The Latrobe Valley.

To refer now to the Latrobe Valley: it is an area of outstanding importance from an industrial viewpoint, as in it exist most of the known fuel sources in Victoria. In an area 20 miles square are proved brown coal supplies which, at our present rate of consumption *per annum*, represent 3000 years' supply.

Very approximately, one-half of Victoria's fuel is used in power and steam raising in industry, one-quarter in gas making, and one-quarter in railways. Most of the fuel used in the first category (power) now comes from brown coal; in the second category, plans for gas from brown coal are already completed and some construction is under way; whilst in the third (railways), research and trials with pulverized brown coal are proving successful. Thus in the next generation the 7,000,000 tons of brown coal burned today should increase three or four times. Whereas the last generation saw the beginning of the liberation of Victoria from the stranglehold of New South Wales coal, the next generation should see its completion.

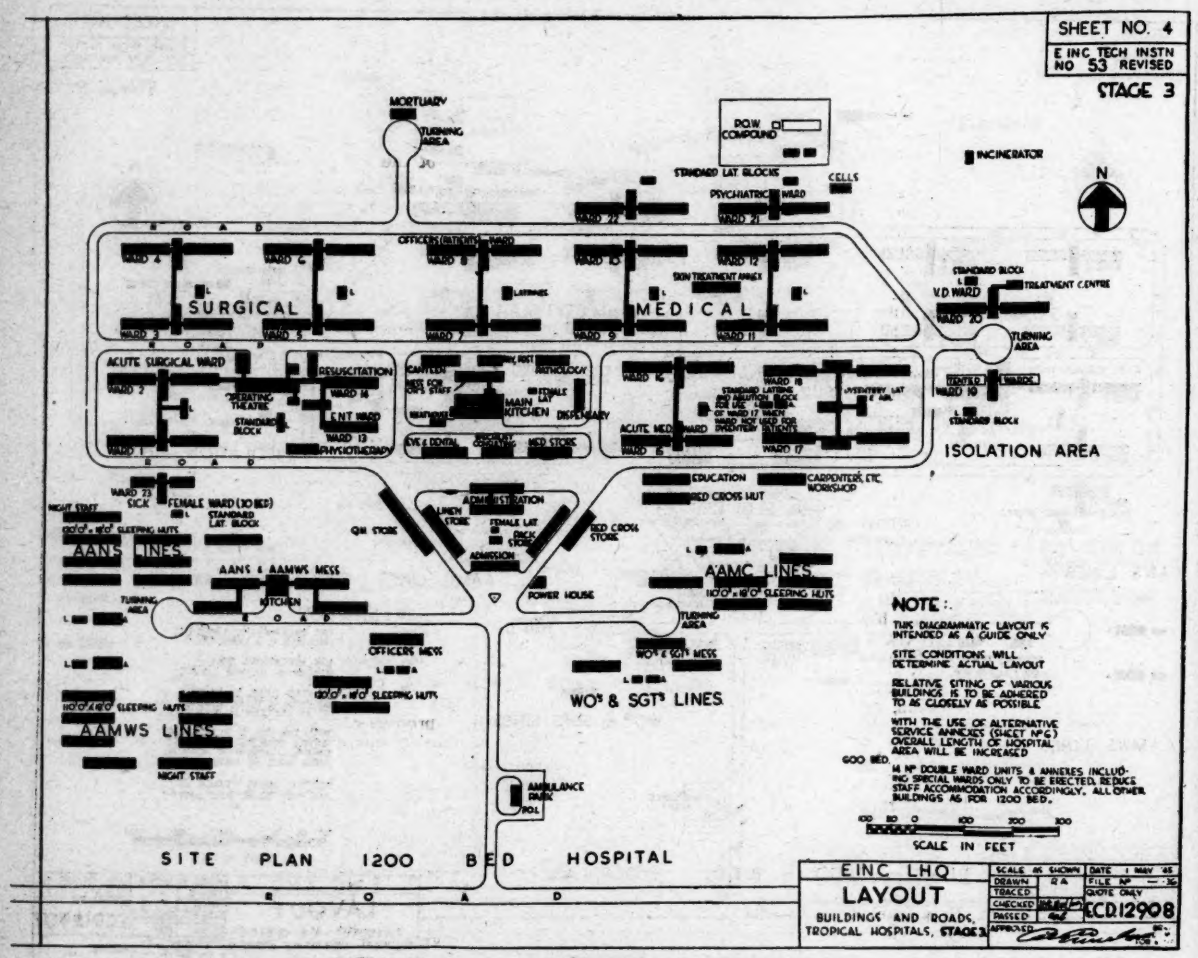


FIGURE III.

Heavy coal-burning industries tend to concentrate round a coal field, because of the cost of transport of fuel. This will be so in the Latrobe Valley, as brown coal, unless made into briquettes, contains one and a half to two tons of water to every ton of combustible matter. The economic limit of distance for carrying raw brown coal is assessed at thirty miles, after which it pays to make it into briquettes. This has made the raw brown coal from Bacchus Marsh the salvation of many industries in Melbourne in these critical years. A new technique has had to be created, that of burning raw brown coal efficiently. This has been achieved basically by the use of the spreader stoker in lieu of the black coal chain grate stoker. If a company has installed spreader stokers, it can then burn most solid fuels. At the present time, as a matter of interest, the comparative costs in Melbourne are in the following proportions: raw brown coal 100, briquettes 116, black coal 142, oil 167. Freight, of course, is a major factor, in the case of raw brown coal being nearly as great as its cost.

To revert to Latrobe Valley industries, these are handicapped by the lack of a port. However, there are the makings of a good port at Welshpool, and I think within this century it, and the adjacent brown coal fields, will be developed.

It should be recalled that much research has gone on during the last century into black coal and its applications. The same to a considerable extent now applies to brown coal. I have mentioned the use of pulverized brown coal in

locomotives; a jet turbine using pulverized brown coal is a practical possibility and may revolutionize industry as much as did the steam engine. The Research Department of the Engineering School of the University of Melbourne has already made coking briquettes in the laboratory. It is thus not altogether a pipe dream to visualize a steel works at Welshpool.

Some of those present may be thinking of uranium. I cannot tell you much about it. Atom plants are the biggest single users of black coal in the United States of America, their planned usage being 22,000,000 tons *per annum*, or one and a half times the total amount of coal used in Australia. So that we may not indulge in too much wishful thinking, I will quote Dr. L. R. Halstad, Director of Reactor Development of the Atomic Energy Commission, United States of America:

> There is still a lot to do to make nuclear fuel competitive with normal fuels, and I am certain nuclear development will not come so fast as to do anything but supplement power from other sources.

However, the main immediate developments in the Latrobe Valley are power, briquettes and gas. The peak winter load of electricity this winter was 550,000 kilowatts; to make things simpler I am going to refer to 100,000 kilowatts, which is about 140,000 horse-power, as one unit. Thus last winter we used power at a maximum rate of five and a half units, of which two units were generated in Yallourn, three in Melbourne, and the remaining one-half of a unit in the various country centres. Kiewa generates one-quarter of a unit. Originally it was planned to produce three units at Kiewa by 1956, but this is now modified to a total of about one unit by then, the balance being deferred more or less indefinitely; the reason is that as there is not enough money available for all, brown coal stations will produce results most quickly and I think most cheaply. I would stress that Kiewa is the only true water power scheme; Eildon and the others generally are storing water in the peak power season, the winter, not creating power.

Yallourn power is, I think, the cheapest we can produce, because obviously the place for the power station is on the coal field, it being cheaper to transmit power by transmission lines than coal by trucks. The present capacity is two units; it is now being increased by 50%, the first half of which should be ready by 1954 and the second by 1955. Our total power production by 1957 will represent an increase over this winter's peak of about 60%. A further site is being investigated for still further extensions.

At Yallourn the first opening was made by the use of power shovels and tip trucks, but it was early decided to adopt methods of overburden stripping and coal winning by the use of dredgers that had been developed with successful results for brown coal open-cut workings in Germany. Bacchus Marsh, with a comparatively small output, still uses shovels at the present time.

Coal in the present open cut at Yallourn has an average thickness of 200 feet and is won by three coal dredgers. Two of these are bucket-chain dredgers, and recently a bucket-wheel dredger has been brought into use. Coal is loaded directly into trucks hauled by electric locomotives. There is an interesting labour-saving machine for track shifting.

The overburden, which has an average thickness of 44 feet, is stripped by two bucket chain dredgers. This machine feeds into trucks, carrying the overburden into the worked-out portion of the open cut, where it is spread over the floor of the open cut by two overburden spreaders.

Briquettes, compressed dried brown coal, are, as we all know, a most useful fuel for many purposes; they have a calorific value equal to 80% of black coal. Unfortunately, they are useless in locomotives. The present briquette factory has an output of 500,000 tons *per annum*. Proposed extensions have a fivefold capacity.

To deal with towns gas, a Lurgi plant is being erected by the Gas and Fuel Corporation at Yallourn. One can generalize and say that the plant is expensive but the fuel is cheap, and the ultimate gas should be no more expensive, unless we have galloping inflation, than at present. It may even be cheaper. The process is a continuous one, and consists of passing steam and oxygen at a high temperature and pressure through briquettes. The resultant is mainly towns gas with a few by-products. One ton of briquettes will produce twice as much gas as one ton of black coal, but with correspondingly fewer by-products. I will endeavour not to bring too many figures into this, but it is anticipated that by 1956 the first stage of the Lurgi plant will be complete (including steam raising and air liquefaction plants), by 1964 it will be quadrupled, and by 1974 it will be doubled again. The first stage will produce approximately half of Melbourne's gas requirements.

I think I can do no better than sum up the future in the words of Dr. Andrews, chairman of the Gas and Fuel Corporation:

> If the Gas Industry in this country puts its shoulder to the wheel it is quite probable that it could, within two to three decades, produce sufficient rich gas, oil and petrol to replace nearly half the liquid fuel now being imported. By such a progressive step this industry could make a valuable contribution to the internal economy and security of the Commonwealth.

In all the foregoing expansions in the Latrobe Valley, a population of 90,000 is being catered for. In addition to the State Electricity Commission, the Gas and Fuel Corporation and private industry, the plans of the State Rivers and Water Supply Commission, the Country Roads Board, the Railways, the Public Works Department (schools and police stations), the Hospitals and Charities Board, several municipal councils, the Housing Commission and others—all have to be integrated. This process has been, since 1949, in the hands of the Latrobe Valley Development Advisory Committee.

Irrigation in the Goulburn Valley.

Whereas the Latrobe River flows south to Bass Strait, the Goulburn, with its origin north of the Divide, flows northerly into the Murray. It is the biggest river in Victoria, but small by world standards. Nevertheless, by any standard the Eildon Dam is a vast project. A contract for £11,000,000 was let to the Utah Construction Company some three years ago. It was the largest single contract ever let in Australia, and Eildon will be the biggest earthen dam in the southern hemisphere. It will be more than half a mile long and will rise to 260 feet above the bed of the river, containing some 13,000,000 cubic yards of earth. It is being constructed immediately below the present dam, and will increase the storage nine times. It will bring into production greater quantities of primary produce in a shorter space of time than any other project in Australia.

Although the water stored is increased ninefold, the area irrigated is only doubled. This is because the water stored has to carry the irrigated area over a possible succession of dry seasons, and also because provision has been made for more intensive irrigation.

The total area in the Goulburn irrigation system is one and a quarter million acres, again the biggest system in Australia, involving 2300 miles of supply channels.

This project when completed (now expected to be in about 1956) will be a tremendous boon to the development of the State.

The following are other notable irrigation projects just being completed: (*a*) the Murray Valley scheme, an area of 270,000 acres supplied by the Yarrawonga Reservoir on the Murray, and north-east of the Goulburn system; (*b*) Robinvale Soldier Settlement, small but highly productive (in four years production has risen from nothing to £500,000); (*c*) the Central Gippsland project, 44,000 acres from the Glenmaggie Reservoir near Maffra and Sale.

In the foregoing I have dealt only with irrigation works; water conservation for stock and the like, in a vast way, is also being carried out by the State Rivers and Water Supply Commission.

Railways, Roads and Ports.

I will deal only very briefly with railways, roads and ports.

We have evidence around us of the new diesel locomotives. Much rolling stock is also being replaced. The Yallourn line is being duplicated and electrified. In the not far distant future the Geelong line will be also. Further ahead, that will also apply to the Ballarat and Bendigo line. For long and big haulages nothing ought to be able to compete with railways.

With regard to roads, one has only to travel over them to know how much has to be done. From Montreal, about the size of Melbourne, to Windsor, about the size of Geelong, a distance of 400 miles, there is being built a two-way express highway—that is, no crossings, no speed limit. A considerable proportion of it is already constructed.

The Country Roads Board had a programme of spending £15,000,000 per year on the roads here. It has been withered by the financial drought; but a start will be made, it is hoped, in the near future, on dual-lane express highways from Oakleigh to Dandenong, and from Footscray to Werribee. All the reconstruction of bridges, deviations *et cetera* in Victoria are being planned and carried out so that ultimately all our main highways will approach the *Autobahn* type.

I have dealt very briefly with roads and railways, but I think everyone should appreciate how much transportation means to the community. In 1949–1950, the latest figures available for Australia, of the total national expenditure, about 40%—£840,000,000 to be exact—was spent on internal transportation. Of this figure approximately 75% was spent on road transportation, 13% on railways, and the remaining 12% on coastal shipping, air transport and tramways.

To deal now with ports, 75% of Victorian exports and imports pass through the port of Melbourne. Every four hours, day and night, a vessel berths at Melbourne, in one of the 105 berths with 60 acres of wharves and 30 acres of transit sheds. In terms of overseas and interstate trade, and also of total shipping tonnage, Melbourne is the busiest port in Australia, handling about 9,000,000 tons of cargo *per annum*.

The development programme of the Melbourne Harbour Trust, now well in hand, includes the following: the building of the River Entrance Docks, where up to 18 liners will be accommodated simultaneously at berthage of the highest world standard; the construction of the modern and mechanized Appleton Dock, west of Victoria Dock, for 18 cargo ships; erection of five new berths along the south wharf of the River Yarra; land reclamation and pier construction at Williamstown, for providing berths for the largest tankers; and the provision of a great deal more mechanical equipment in a port which is already the most highly mechanized in Australia.

Where is the money to do all these things to come from?

I feel I am on sacrosanct ground here; but I trust that the fundamental truth will be recognized in time by all—that the wealth of a country is the work of its people and the more mechanized they are the greater will be their output, the cheaper the goods, and the greater the purchasing power and therefore ultimately the wealth of the community.

In England and here, priority of effort has been given to housing, in Germany to factories. While our outlook is more humane, it remains to be seen, after a decade, whether the respective peoples may not be better off by doing things the hard way. The following has been extracted from a recent article:

> All over Western Germany, far into the night by the light of arc lamps and at the first crack of dawn, reconstruction goes on. All over Germany the men and women are working with a will, as busy as beavers. Taking the index of industrial production in 1936 as 100, production has been pushed from a mere 36·1 in 1948 to 156 in May of this year. Today the Western mark is one of the world's hardest currencies. It is as sought-after as the dollar or the Swiss franc.

Hospitals.

I consider that this address would be incomplete without reference to hospitals.

During the war in the South-West Pacific Area, we could build a fully hutted, 1200 bed hospital in eighteen weeks. True, the roof was iron, the walls were fibreen (sacking impregnated with bitumen), all unlined. You will be interested to know that the plans were prepared by some of the best hospital architects, temporarily in the Royal Australian Engineers, working in conjunction with the senior medical officers of the Army.

I am not suggesting that a temporary building on the Equator is suitable for our conditions here, but I do suggest that we should not put too much of our effort into work that is productive only to a limited extent, though it is very essential.

The State Rivers and Water Supply Commission is very proud of its hospital at Eildon, a township of 4000 inhabitants. Some old Army huts were bought cheaply. I am told that it cost £17,000 to make this 17 bed hospital.

Whilst some big hospitals are essential, a number of decentralized smaller ones on the fringe of the suburbs may be economical. The advantages of the big hospital are centralized services *et cetera*; the advantages of the smaller hospital are more personal interest in and from the surrounding community, and gardens in place of cantilever verandas; large halls and expensive lifts become unnecessary, and costly framed construction can be replaced by comparatively inexpensive single-storied buildings.

Conclusion.

In conclusion, I should like to say that we have inherited a tremendous responsibility as well as a tremendous asset. It is our duty to develop this land with all expedition by work, by mechanization, and by rapid immigration, even at the risk of temporary inconvenience. Otherwise someone else may do it. Finally, let me quote the words of Mr. T. N. Dougherty, Federal Secretary of the Australian Workers' Union:

> . . . but it's a great country, the best in the world today. It's got a great future if we work for it.

EARLY EFFECTS OF LEAD ON LYMPHOID CELLS.

By D. O. SHIELS,
Industrial Hygiene Division, Department of Health, Victoria.

IN previous papers (Shiels, 1936, 1937, 1950), I have confirmed the findings of Ferguson and Ferguson (1934) that the ratio of monocytes *plus* large lymphocytes to small lymphocytes is increased in persons who have absorbed significant amounts of lead, and remains at an increased value so long as the absorption of lead is not of too great an amount for too long a time, and further, that this ratio is more closely related to the clinical condition than is the stippled cell count.

Lane (1949) has stated that the changes in the ratio are not sufficiently constant to be of practical value.

It might be thought that the difference of opinion was due to differences of technique in making or staining the film, or in the regions in which the ratio was determined.

I have determined the ratio in over 2300 blood films of a variety of subjects, including those not exposed to any occupational hazard, those exposed to lead hazards but without symptoms, those suffering from lead poisoning, those who are recovering from lead poisoning, and those who have recovered from lead poisoning. I am convinced of the usefulness of this test in the prevention and diagnosis of lead poisoning.

However, it is not the purpose of this paper to discuss these points, but to indicate that, although the ratio may be different in different parts of a film, if a particular technique is always carried out, results which are sufficiently consistent to be of great value can be obtained, and secondly, to show how early is the response of the lymphocytes to lead absorption.

The present paper records the results obtained in the case of five persons at the beginning and end of their first week of exposure to lead.

In this investigation, two ratios have been determined—namely, the ratio of monocytes *plus* large lymphocytes to small lymphocytes $\left(\frac{L+M}{S}\right)$, and the ratio of large lymphocytes to small lymphocytes $\left(\frac{L}{S}\right)$.

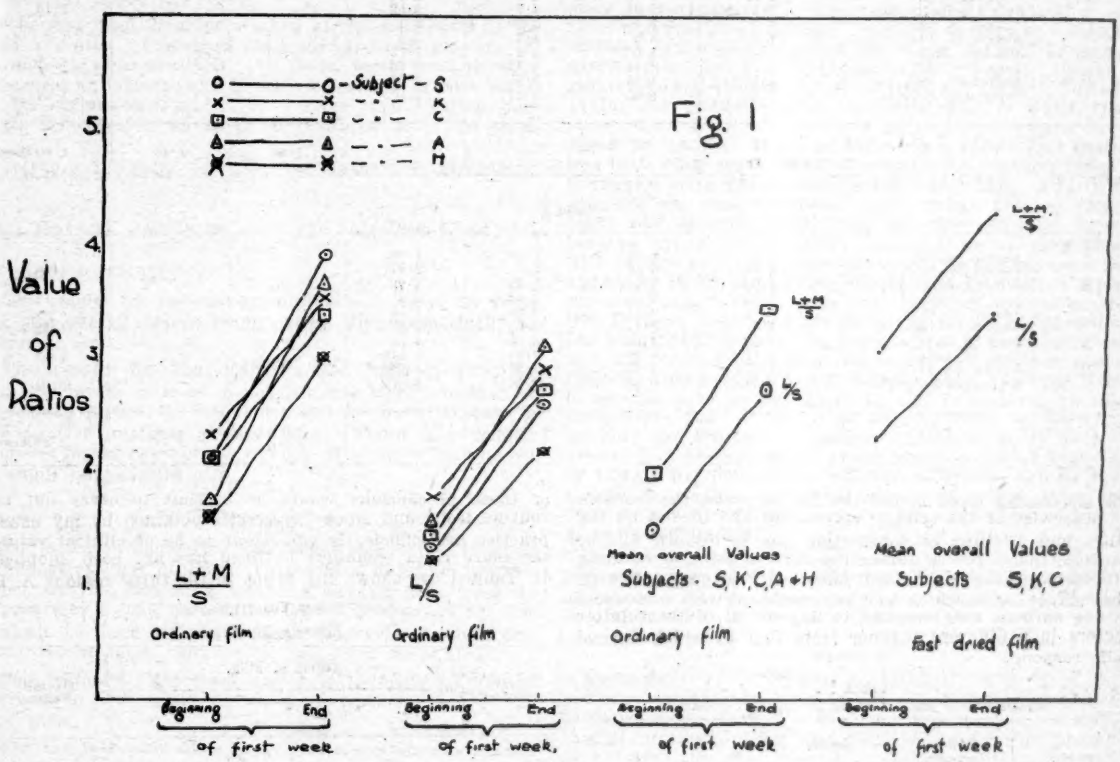


FIGURE I.

Although in the case of both $\frac{L+M}{S}$ and $\frac{L}{S}$ the ratios determined in three regions of each film differ from one another (that is, at the beginning, the middle and the end), they had in all but one of the 15 determinations increased at the end of the first week of exposure by a significant amount for each region of the films.

At the end of their first week of employment the concentrations of lead in the urine of the five subjects were 0·09, 0·15, 0·11, 0·08 and 0·15 milligramme per litre respectively.

The technique which I usually employ is as follows. The blood is spread carefully so as to leave a film one cell thick, and finishing with "tails", not with a thick line of blood at the far end. After being dried in air, it is stained for 1·5 minutes with Leishman's stain (0·1 gramme per 10 cubic centimetres of acetone-free methyl alcohol). The stain is then diluted with about double the volume of water and left for ten minutes. The film is then quickly washed and dried, either by being drained in air or with blotting paper. Microscopic examination is carried out with an oil immersion lens at a magnification of 900. The large and small lymphocytes are counted in strips at right angles to the long axis of the film and extending from edge to edge of the film. The large cells are taken arbitrarily to be those greater than 10μ in diameter, the size being determined by comparison with a linear scale or a squared grid inserted into the ocular of the microscope.

The regions are designated as follows: near end, B; middle, A; far end "tails", C. About 30 lymphocytes and monocytes are counted in each of the regions A and B, and about twice this number in C, and the ratio of the aggregate number of large cells in A+B+C to the aggregate number of small cells in the same regions is taken as the overall ratio. This overall ratio is the one used for diagnostic and clinical purposes.

The reason for counting a larger number of cells in the tails is that, as usually made, the film shows relatively considerably more monocytes in the tails than in the other regions—indeed, a greater number of all white cells per unit area of film. Since there is a differential distribution of white cells along the film, it is obviously not strictly satisfactory to exclude any part of the film in the count.

However, in the extreme ends of the "tails" the cells are often so broken up and distorted that it is not possible to assess their sizes accurately. Therefore, the extreme ends of the tails are omitted when this is the case. Thick films are quite unsatisfactory.

The factors which may influence the sizes of the cells in the film are, of course, the actual size of the cells in

TABLE I.

Ratios $\frac{L+M}{S}$ and $\frac{L}{S}$: Ordinary Film.

Subject.	Date of Test.	Region of Film. A.	Region of Film. B.	Region of Film. C.	Overall Value.	Mean Overall Value. At Beginning of First Week.	Mean Overall Value. At End of First Week.	Increase in Overall Value at End of First Week.
				Ratio $\frac{L+M}{S}$				
S.	August 8, 1951.	3·4	2·4	2·4	2·1			
	August 15, 1951.	4·5	3·66	3·7	3·9			1·8
K.	July 11, 1951.	2·66	2·08	2·5	2·39			
	July 18, 1951.	3·8	2·25	4·7	3·52			1·13
C.	August 8, 1951.	2·0	2·4	2·06	2·1			
	August 15, 1951.	2·6	3·0	4·6	3·4			1·3
A.	June 20, 1951.	1·44	1·0	3·2	1·73			
	June 27, 1951.	3·33	3·44	4·125	3·60			1·87
H.	August 29, 1951.	1·15	1·64	1·83	1·58			
	September 5, 1951.	1·93	2·63	4·13	3·0			1·42
						1·98	3·48	1·50 Mean
				Ratio $\frac{L}{S}$				
S.	August 8, 1951.	2·4	1·09	1·21	1·39			
	August 15, 1951.	2·66	3·16	2·0	2·77			1·38
K.	July 11, 1951.	2·55	1·7	1·32	1·78			
	July 18, 1951.	3·33	2·0	3·43	2·9			1·12
C.	August 8, 1951.	1·75	2·17	0·88	1·51			
	August 15, 1951.	2·2	2·7	3·6	2·83			1·32
A.	June 20, 1951.	1·18	1·00	3·00	1·59			
	June 27, 1951.	3·11	3·33	2·88	3·115			1·53
H.	August 29, 1951.	1·077	1·43	1·277	1·266			
	September 5, 1951.	1·47	2·25	2·875	2·205			0·94
						1·51	2·77	1·26 Mean

the circulating blood before the film is made, the freedom or otherwise of the cells to spread out and lie flat on the slide, and swelling or contraction due to osmotic and/or surface tension forces during the drying, fixing or staining processes. Cells which have adsorbed lead compounds on the surface, or in which lead has combined with substances in the surface, may respond to any or all of these latter factors in a different manner from that in which normal cells respond.

TABLE II.

Significance of Mean Increase in Ratio, Five Subjects.

Ratio.	Mean Increase.	Value of *t*.	*n*.	*P*.
$\frac{L+M}{S}$	1·50	10·47	4	<0·001
$\frac{L}{S}$	1·26	12·20	4	<0·001

Apparently, rapidity of drying affects the size of the white cells in the film, there being frequently relatively fewer large cells in those parts which dry out last. A film spread on a warmed slide so that it dries out rapidly will have a higher ratio than one allowed to dry in the ordinary way. However, the effects of exposure of persons to lead are in the same general direction in the case of their quickly dried films as in the case of their ordinary films.

Possibly the ideal method of making a film would be to use a drop of blood of definite size measured by blood pipette, placed on a slide at a definite temperature, exposed to air of definite temperature and humidity, and spread mechanically by a spreader adjusted at a fixed angle and time-controlled to move at a fixed rate. There would then still be some differential distribution of the cells along and across the film due to difference in sizes or weight, or surface tension effects, and "leaded" cells might respond differently from the way in which normal cells do. Certain of these precautions would be difficult to carry out in routine tests, and since the results obtained by my usual practice are sufficiently consistent to be of clinical value, the more rigid technique outlined has not been adopted. In Table I are shown the ratios in the three regions A, B and C for the five persons at the beginning and end of their first week of exposure to lead, and the ratios determined from the total counts in A+B+C (the overall ratio).

TABLE III.

Film Rapidly Dried.

Subject.	Region of Film. A.	Region of Film. B.	Region of Film. C.	Overall Value.
		Ratio $\frac{L+M}{SS}$		
S.	3·0	2·3	5·1	3·2
	3·9	7·0	2·75	4·26
K.	4·6	4·2	3·5	3·9
	17·0	>26·0	1·9	4·52
C.	7·5	5·7	1·63	2·1
	3·5	6·16	3·7	4·24
		Ratio $\frac{L}{S}$		
S.	2·72	1·75	3·66	2·53
	8·3	6·25	1·93	3·52
K.	4·2	3·6	2·17	2·95
	15·0	>25·0	1·0	3·84
C.	6·5	4·3	1·16	1·67
	3·12	2·12	2·54	3·44

In Figure I the results are shown diagrammatically. With the ordinary films there was a definite increase in the ratio $\frac{L+M}{S}$ at the end of the first week in each case in

each region, and, of course, also in the overall values. Out of the 15 values for the ratio $\frac{L}{S}$ in the different regions at the end of the first week, only one showed a slight decrease from the value shown at the beginning of the week. In all five cases the overall ratio $\frac{L}{S}$ showed an increase at the end of the first week over the values found at the beginning.

The increases in the overall values of the two ratios were statistically significant.

Table II shows the results.

The fast-dried films were taken at the same time as the ordinary films. Fast-dried films of only three subjects (S, K and C) were available. In these, there were in some instances an increase and in some a decrease in both ratios in the regions A, B and C, but for the overall ratios there were increases in all cases for both ratios. The mean increase in the ratio $\frac{L+M}{S}$ was not statistically significant, but that in the ratio $\frac{L}{S}$ was significant ($n=2{\cdot}0$, $t=4{\cdot}407$, $P<0{\cdot}05$).

The results for the fast-dried films are shown in Table III, the overall values being shown diagrammatically in Figure I.

The results for the ordinary film indicate that the increase in the ratio of monocytes *plus* large lymphocytes to small lymphocytes is not due merely to an increase in the relative numbers of monocytes. There is a definite increase in the ratio of the number of large to the number of small lymphocytes also.

Summary.

The ratios of monocytes *plus* large lymphocytes to small lymphocytes, and of large lymphocytes to small lymphocytes, at the beginning and end of the first week of exposure to a lead hazard, have been determined for five subjects in three regions of the ordinary blood films, and for three of these subjects for fast-dried films also.

For ordinary films there was a statistically significant increase in both ratios for all subjects at the end of the first week.

For the fast-dried films there was a statistically significant increase in the ratio of large to small lymphocytes.

The results indicate a very early effect of lead on the lymphoid cells.

Acknowledgements.

I wish to make acknowledgement to Dr. A. J. Christophers, B.Sc., for his interest in this work, and for useful discussions, and to Mr. F. N. Hutchison for drawing the diagram.

References.

FERGUSON, A. E., and FERGUSON, T. (1934), "Examination of Blood Films in Relation to the Prevention of Plumbism among Shipbreakers", *J. Hyg.*, 34: 295.

LANE, R. E. (1949), "Care of the Lead Worker", *Brit. J. Indust. Med.*, 6: 125.

SHIELS, D. O. (1936), "The Ratio of Large to Small Lymphocytes in Persons Exposed to a Lead Hazard", *M. J. AUSTRALIA*, 1: 539.

SHIELS, D. O. (1937), "Comparison of Punctate Basophilia and Ratio of Large to Small Lymphocytes in the Diagnosis of and Prevention of Lead Poisoning", *M. J. AUSTRALIA*, 1: 535.

SHIELS, D. O. (1950), "Influence of Lead Absorption on the Ratio of Large to Small Lymphoid Cells", *M. J. AUSTRALIA*, 2: 205.

THE ASSOCIATION BETWEEN HYPOGLYCÆMIA AND MYOCARDIAL INFARCTION.

By BRYAN GANDEVIA,
Royal Melbourne Hospital, Melbourne.

If imputation and strong circumstance—
Which lead directly to the door of truth—
Will give you satisfaction, you may have 't.

—*Othello*, Act iii, Scene 3.

THE introduction of a new and powerful therapeutic agent is usually followed by the appearance in the literature of "cautionary tales" which it may take some years to place in perspective; in this respect the introduction of insulin proved no exception. A number of cases of *diabetes mellitus* were soon reported in which *angina pectoris*, cardiac infarction or sudden death (Parsonnet and Hyman, 1931) followed the institution of insulin therapy. Turner (1930) and Modern (1931) described cases in which the onset of cardiac pain was related to hypoglycæmia produced by insulin. Study of these cases shows that there was little other than "imputation and strong circumstance" to suggest more than a coincidental relationship. A further complication was introduced when Sippe (Sippe, 1933; Sippe and Bostock, 1933) reported cases of mild spontaneous hypoglycæmia which appeared to be associated with cardiac pain. Although in the last decade advances in knowledge of the clinical and biochemical features of hypoglycæmia have brought fuller understanding of the association between it and cardiac pain, the precise relationship has remained ill defined. No unanimity as to its importance is to be found in standard text-books, the opinions given ranging from a warning that hypoglycæmic reactions may cause angina or sudden death in elderly diabetics to the observation that deaths from hypoglycæmia in *diabetes mellitus* are clinical curiosities. Since coronary artery disease and hypoglycæmia, either spontaneous or induced by insulin overdosage, are common disorders, and as the former is usually capable of control, it is important to decide whether coincidence of the two conditions is likely to prove detrimental.

The present paper aims to assess the frequency of this association, to present some further evidence on its possible significance and to summarize recent published work on the relationship between hypoglycæmia and cardiac pain.

Frequency.

Assessment of incidence is primarily dependent on accuracy of diagnosis. To exclude error in regard to the cardiac lesion, study has been confined to cases of myocardial infarction of less than three weeks' duration proven at autopsy. In the absence of absolute criteria for the diagnosis of hypoglycæmia it can only be stated that the diagnosis was not accepted without sound clinical or biochemical evidence.

In a series of 50 consecutive cases of myocardial infarction six patients were found to have had an antecedent hypoglycæmic reaction in approximately appropriate time relationship to the time of onset of the infarct as determined from the history and the histological examination of the specimen. Five of these patients were diabetics who had been receiving insulin for some years, and the sixth was found to have an islet-cell tumour of the pancreas. The clinical history of the last-mentioned patient is cited because of its particular interest and because it may appear as an exception to the criteria laid down above.

CASE I.—The patient was a male, aged sixty-six years. Eight years prior to his final admission to hospital he suffered from aplastic anæmia due to benzol poisoning, from which he made a complete recovery, subsequent blood examinations over the years revealing no abnormality. Six months later he complained that he was still unfit for work because of attacks of sweating, "blackouts" and "weakness all over"; at this time he was noted to be becoming more introspective. Late in 1945, a year after his first illness, complaints of constricting and suffocating sensations in his neck were regarded by the psychiatrist as compatible with the diagnosis of anxiety neurosis and depression. In 1947

he developed tremor of his hands, particularly when attempting delicate work; he was considered to be suffering from neurosis and hysteria. One year later he suffered from pain closely resembling that of *angina pectoris*, the attacks being associated with breathlessness, but occurring both at rest and on exertion. There were no significant changes in an electrocardiogram and the diagnosis appears to have been uncertain. He remained weak and unable to hurry until 1951, when there are notes of several faints and "collapses" (notably at a time of domestic stress), cold sweats at night and "pains round the heart". His relatives, questioned after his death, stated that the pains in the chest occurred chiefly in the afternoon between meals, were related to exertion, and were usually but not always relieved by rest within a quarter of an hour. For many years his diet had been one of low protein and relatively high carbohydrate contents. When he was admitted to hospital in December, 1952, with a typical history of myocardial infarction, he is recorded as stating that he had had severe chest pain related to exertion and relieved by trinitrin for about a year. He was described as rubicund and healthy-looking; his blood pressure was 200 millimetres of mercury, systolic, and 110 millimetres, diastolic, the readings eight years previously having been 135 and 80 millimetres of mercury respectively. He died suddenly shortly after his admission to hospital. At autopsy he was found to have gross coronary atherosclerosis, coronary thrombosis with infarction and a carcinoma of the tail of the pancreas, in some areas of which islet tissue was readily recognizable. In retrospect his private physician considered that hypoglycæmia was the probable cause of his attacks, and this would also account for the mental changes, the tremor and the atypical anginal pain.

Three further case summaries may be taken as representative of the remaining cases.

CASE II.—The patient was a female, aged fifty-nine years, a diabetic, who had been treated with insulin for seven and a half years; she was admitted to hospital with a fractured pelvis and senile dementia. Her diabetic condition proved difficult to stabilize during her lengthy illness owing to a persistent urinary infection and, during the last week of her life, bronchopneumonia. She had hypoglycæmic episodes on the seventeenth and eighteenth days before her death. At autopsy a cardiac infarct between two and three weeks old was found; the coronary arteries, although thick-walled, were patent.

CASE III.—The patient was a female, aged sixty-eight years, a known diabetic, who had been treated with insulin for seventeen years. She was admitted to hospital after five days of an illness characterized by vomiting, anorexia and polyuria, with progressive impairment of consciousness over the previous twelve hours. She had taken no insulin for two days and was in a state of severe diabetic acidosis with peripheral circulatory failure. An electrocardiogram showed rapid auricular fibrillation and a left bundle branch block to be present. A quarter of an hour after 200 units of insulin had been given intravenously, the blood sugar level was 410 milligrammes *per centum*, and this was reduced over twelve hours to less than 100 milligrammes *per centum*. With other restorative measures her condition greatly improved, although an associated minor cerebral thrombosis was suspected chiefly because of the tardy improvement in her mental state. On the morning of the third day in hospital the blood sugar level was 20 milligrammes *per centum*, and on the sixth day she showed signs of hypoglycæmia. She appeared relatively well on the tenth day, except for some continued impairment of her mental faculties, but she had a further hypoglycæmic episode during the morning. She died in the evening, shortly after a sudden deterioration in her condition. At autopsy an extensive infarct involving the left ventricle was found; the coronary arteries were moderately atheromatous, but showed no obstruction. On histological examination an infarct twelve to fourteen days old was associated with areas of more recent fibre necrosis and early leucocytic infiltration.

CASE IV.—A male patient, aged forty years, a known diabetic, had been receiving insulin for twelve years. He was admitted to hospital for treatment of intractable ulceration of one foot and was given 25 milligrammes by mouth of "Priscol" every four hours. He required the intravenous administration of glucose to relieve a hypoglycæmic reaction of moderate severity. Twenty-four hours later he collapsed suddenly and died within three hours. In addition to the scar of an old infarct, a very recent thrombus was found in the left coronary artery with adjacent recent infarction.

It is stressed that the figure of 12% thus obtained for the coincidence of hypoglycæmia and cardiac infarction relates merely to this coincidence; no claim that the hypoglycæmic episode precipitated the infarction can be substantiated on evidence of this kind. However, an approximate estimate of the frequency of the association allows it to be viewed in better perspective than is permitted by the recording of individual selected cases. It appears to have escaped comment that there are valid reasons for not insisting on strict sequence and accurate time relationships before the association can be considered of serious significance. The chief of these are as follows:

1. The hypoglycæmic phase may act as an aggravating rather than as a precipitating or determining factor.

2. In an uncomplicated hypoglycæmic episode, signs of a hyperdynamic circulatory state and certain changes in the electrocardiogram (Messinger, 1939) may persist for hours or days after the blood sugar level has returned to normal, indicating the persistence of an abnormality beyond the time required for full mental recovery.

3. The onset of pain or collapse may not coincide with the time of onset of the infarct. Such a discrepancy is occasionally noticed at autopsy, while twice recently a pericardial friction rub has been found to develop in patients under observation for unrelated conditions some hours prior to the onset of the symptoms of cardiac infarction.

No consecutive series based on autopsy material appears to have been reported, but figures derived from clinical experience are available for the incidence of a significant hypoglycæmic factor in cardiac disease. Harrison and Finks (1943) thought it important in 31 of 204 consecutive cases of cardio-vascular disease (approximately 15%). In a series of 77 cases of *angina pectoris*, Harrison (1944) found two patients whose attacks appeared to be caused by hypoglycæmia alone, no evidence of structural cardiac disease being demonstrable. In 34 cases the relationship of the pain to food, the failure of rest to produce speedy relief, and improvement on the ingestion of a protein-rich diet suggested that hypoglycæmia might be one factor involved in the production of the attacks. Both papers report the results of clinical experiments to support their conclusions. Parsonnet and Hyman (1931) found 22 diabetic subjects in a series of 89 cases of coronary thrombosis, and in seven of these the coronary episode was closely related to the initial phase of insulin therapy. In all the diabetic patients in the present series insulin had been given for some years.

Significance of the Association in Diabetic Subjects.

The hypothesis that hypoglycæmia may precipitate or aggravate cardiac infarction would rest on a surer foundation if it was shown that hypoglycæmia was associated with death from myocardial infarction more frequently than it was from other natural causes. The present investigation was limited to diabetic patients, so that the decision as to whether hypoglycæmia had been present was usually easily made; only infarcts of less than three weeks' duration and proven at autopsy were accepted.

In an unselected group of 55 diabetic subjects reaching autopsy, 14 were found to have myocardial infarcts; six of these patients had had a hypoglycæmic reaction at approximately the time of onset of the infarct. Forty-one patients died of other causes, four of them having had a hypoglycæmic episode which could have been connected with death or with the onset of the disease leading to death (Table I). One of these four subjects was a male, aged forty years, suffering from *diabetes mellitus* and œdema secondary to renal involvement, but without hypertension. He succumbed to acute pulmonary œdema two hours after apparently complete recovery from a moderately severe hypoglycæmic reaction. As no infarct was demonstrated, and as an extracardiac cause for the pulmonary œdema could not be excluded, his case is included in the control series. However, a similar case in which electrocardiographic evidence of infarction was found has been described by Grainick (1944; quoted by Gilbert *et alii*).

The difference in incidence of antecedent hypoglycæmia between the two groups is significant, and supports the contention that hypoglycæmia bears more than a casual relationship to myocardial infarction. No comparable overseas figures have been found.

Discussion.

If the relationship of hypoglycæmia to myocardial infarction is accepted as significant, then the more common problem of a relationship to cardiac pain arises. Until the last decade the literature is confusing on this point, probably chiefly because of difficulty in distinguishing three different sets of circumstances, all related to lowering of the blood sugar level, in which chest pain may occur. In addition, some earlier reports are open to reassessment in the light of recent advances in knowledge of the symptomatology and physiology of the hypoglycæmic state.

The first set of circumstances is the commencement of insulin therapy in patients with *diabetes mellitus*, which has been followed by the development of *angina pectoris* or myocardial infarction or by sudden death (Parsonnet and Hyman, 1931) in the absence of any clinical evidence of hypoglycæmia. Although Soskin *et alii* (1933) found

TABLE I.[1]

Subjects.	Antecedent Hypoglycæmia Present.	No Antecedent Hypoglycæmia.	Totals.
Subjects with myocardial infarcts	6	8	14
Subjects without myocardial infarcts ..	4	37	41
Totals	10	45	55

[1] There was no significant difference between the average age at death of the hypoglycæmic group and the group without antecedent hypoglycæmia (63±11 years). There was also no difference between this series and the 50 unselected cases of myocardial infarction in regard to age. The sex incidence showed no unusual features, females predominating in the above groups.

subjective and objective evidence of impaired cardiac function during insulin treatment compared with periods of dietary control alone, they obtained the same result in patients treated without insulin but with diets of high or low carbohydrate content, so that a "toxic effect" of insulin is unlikely to be responsible. On the whole, the evidence for the existence of this group of cases rests on the description of isolated cases and can only be regarded as inconclusive. Even if the association occurs more frequently than can be accounted for by chance, there would seem to be no reason for considering such cases as fundamentally any different from those in which the fall in blood sugar level is sufficiently great or sufficiently rapid to produce the symptoms of a hypoglycæmic reaction. No case which could be considered as belonging to this group was encountered in the present series.

The second set of circumstances comprises those cases in which clinical or perhaps biochemical evidence of hypoglycæmia is associated with the development of the same cardiac disorders. The clinical and experimental evidence for this concept has been presented by Harrison and Finks (1943) and by Harrison (1944), and the possible mechanisms involved have been reviewed by Gilbert *et alii* (1946). Lowering of the blood sugar level *per se* is not solely responsible for the effects observed. The persistence, after the blood sugar level has returned to normal, of the signs of a hyperdynamic circulatory state and of abnormal electrocardiographic findings has already been noted (Messinger, 1939; Middleton and Oatway, 1931); these signs are not always reversed by intravenous glucose. There is a lack of correlation between the blood sugar level and the clinical picture, not only over a group of patients, but also in successive observations on the same subject (Soskin *et alii*, 1935), while symptoms are more easily produced if the rate of fall is rapid. It has also been pointed out that the heart is not directly dependent on circulating glucose for its metabolism, and that the high glycogen content of the diabetic heart falls only to normal during hypoglycæmia. Finally, Gilbert and his co-workers have shown that the circulatory changes of hypoglycæmia are inhibited by a parasympathomimetic drug such as "Prostigmin". Since these changes are also abolished by combined denervation of the heart and adrenalectomy, but not by either procedure alone (Cannon *et alii*, 1924), it has been suggested that the circulatory phenomena are the result of sympathetic stimulation. The development of *angina pectoris* or myocardial infarction and the electrocardiographic changes are thus viewed as an expression of increased cardiac work in the presence, in all probability, of some coronary sclerosis. This is regarded as "the most tenable theory" of their causation (Liebow and Hellerstein, 1949), but although it is adequate for clinical purposes it probably represents an over-simplification of the problem.

The final set of circumstances in which hypoglycæmia may be associated with cardiac symptoms and chest pain is represented by cases of cardiac neurosis. The hypoglycæmia is more commonly spontaneous or functional, but symptoms can equally well occur in diabetics receiving insulin. There is little doubt that this group of cases has been responsible for considerable confusion in the past. Sippe (Sippe, 1933; Sippe and Bostock, 1933) is regarded as the first to describe *angina pectoris* in association with spontaneous hypoglycæmia, but it is probable that most of his examples were cases of the effort syndrome produced or accentuated by spontaneous hypoglycæmia. Nevertheless, much credit is due to Sippe for his insistence on the relationship of the chest pain to hypoglycæmia in the face of criticism on two counts. Firstly, he was prepared to accept as confirmatory evidence of the clinical diagnosis fasting blood sugar levels which were not considered low enough—for example, 70 milligrammes *per centum*. Secondly, it was suggested that the pain described was more like that of a functional disorder than true angina. In regard to the first objection, it is now appreciated that fasting blood sugar estimations are unreliable in the diagnosis of hypoglycæmia, particularly of the functional variety. In regard to the second objection, the position was clarified by Harrison and Finks (1943) and by Greene (1944). Greene described typical cases of the effort syndrome in young servicemen caused by spontaneous hypoglycæmia, responding dramatically to a protein-rich diet. The symptoms of cardiac neurosis must be accepted as one feature of repeated hypoglycæmic reactions. The difficulty which this may produce in differential diagnosis is well illustrated by Case I above, and also by diabetic patients in whom it complicates organic heart disease.

It may be concluded that there is sufficient evidence to indicate that hypoglycæmia may act, probably indirectly, as a precipitating or aggravating factor in the production of myocardial infarction and *angina pectoris*. On this basis it would seem rational to ensure a protein-rich diet for those patients whose angina is more easily produced or less quickly relieved by rest at times of the day when the blood sugar level is likely to be low. This procedure also seems to serve as a therapeutic test; biochemical tests may be equivocal and may not be entirely devoid of risk. Elderly diabetic patients, especially those with evidence of decreased cardiac reserve, should be managed with particular care to avoid hypoglycæmic reactions. Gilbert *et alii* (1946) have suggested that small doses of "Prostigmin" may be useful in these cases, and at least this would appear to be a justifiable addition to the treatment of the established hypoglycæmic attack.

Summary.

1. In a consecutive series of 50 cases of recent myocardial infarction proven at autopsy, six subjects were found to have had an antecedent hypoglycæmic episode at approximately the time of onset of the infarct.

2. In a series of 55 diabetic patients who came to autopsy, the incidence of antecedent hypoglycæmia was found to be significantly higher in those who had died from myocardial infarctions than in those who had died from other causes.

3. The literature is summarized, and the conclusion is reached that hypoglycæmia, probably acting indirectly by producing an increase in cardiac work, may be a factor in the production of myocardial infarction, *angina pectoris* and some cases of cardiac neurosis.

Acknowledgements.

My thanks are due to Dr. J. H. Bolton and Dr. Pincus Taft for their advice in the preparation of this article, to Dr. F. Blois Lawton for valuable help in regard to a patient under his care, and to Dr. J. D. Hicks for assistance with the autopsy material.

References.

CANNON, W. B., MCIVER, M. A., and BLISS, S. W. (1924), "Studies on the Conditions of Activity in Endocrine Glands", *Am. J. Physiol.*, 69: 46.

GILBERT, R. A., and GOLDZIEHER, J. W. (1946), "The Mechanism and Prevention of Cardiovascular Changes due to Insulin", *Ann. Int. Med.*, 25: 928.

GREENE, R. (1944), "Cardiac Neurosis as a Manifestation of Hypoglycæmia", *Lancet*, 2: 307.

HARRISON, T. R., and FINKS, R. M. (1943), "Glucose Deficiency as a Factor in the Production of Symptoms Referable to the Cardiovascular System", *Am. Heart J.*, 26: 147.

HARRISON, T. R. (1944), "Clinical Aspects of Pain in the Chest—Angina Pectoris", *Am. J. M. Sc.*, 207: 561.

LIEBOW, I. M., and HELLERSTEIN, H. K. (1949), "Cardiac Complications of Diabetes Mellitus", *Am. J. Med.*, 7: 660.

MESSINGER, E. (1938), "Cardiovascular Changes Associated with Insulin Shock Treatment", *Ann. Int. Med.*, 12: 853.

MIDDLETON, W. S., and OATWAY, W. H., JUNIOR (1931), "Insulin Shock and the Myocardium", *Am. J. M. Sc.*, 181: 39.

MODERN, F. S. (1931), "Chronic Heart Pain due to Prolonged Hypoglycæmia", *J.A.M.A.*, 96: 357.

PARSONNET, A. E., and HYMAN, A. S. (1931), "Insulin Angina", *Ann. Int. Med.*, 4: 1247.

SIPPE, C., and BOSTOCK, J. (1933), "Hypoglycæmia: A Survey and Account of 25 Cases", M. J. AUSTRALIA, 1: 207.

SIPPE, C. (1933), "Hypoglycæmic Angina, with a Report of Five Cases", M. J. AUSTRALIA, 2: 302.

SOSKIN, S., KATZ, L. N., STROUSE, S., and RUBINFELD, S. H. (1933), "Treatment of Elderly Diabetic Patients with Cardiovascular Disease", *Arch. Int. Med.*, 51: 122.

SOSKIN, S., KATZ, L. N., and FRISCH, R. (1935), "The Dual Nature of the Action of Insulin upon the Heart", *Ann Int. Med.*, 8: 900.

TURNER, K. B. (1930), "Insulin Shock as a Cause of Cardiac Pain", *Am. Heart J.*, 5: 671.

DISSEMINATED NEURODERMATITIS AND CATARACT.[1]

By R. HERTZBERG, F.R.A.C.S.,
Sydney.

A NOT uncommon dermatological disorder is *neurodermatitis disseminata*, also known as allergic eczema, atopic dermatitis, pruritus with lichenification, eczematoid dermatitis, food eczema and infantile eczema (Andrews, 1946). Many authors regard the condition as atopic since the introduction of this term by Coca (1934).

Definition of Atopy.

Atopy is that form of allergy which, at present, appears to be peculiar to man, which is influenced by inheritance, and which among its other attributes is characterized by the common occurrence of certain specific circulating antibodies. The antibodies have been called atopic reagins (Coca, 1934). The atopic state is characterized by the frequent presence of some of the following criteria: (i) A complex of symptoms and phenomena which can be explained primarily by increased vascular permeability. (ii) A family history of asthma, hay fever, atopic dermatoses and perhaps certain other diseases, such as migraine, hives and gastro-intestinal manifestations. (iii) A personal history which often reveals, in addition to the presenting atopic disease, certain other atopic manifestations. (iv) Eosinophilia in the blood and/or the fluids and tissues of the affected parts. (v) A positive reaction to a scratch or subcutaneous skin test. (vi) Relief upon avoidance of the offending substances (but relief does not always follow avoidance of agents producing positive results with scratch tests). (vii) Recurrence or exacerbation on exposure (Sulzberger, 1940).

[1] Read at a meeting of the Royal Australasian College of Surgeons, Sydney, 1953.

Beetham (1940), who reviewed the literature on atopic cataracts and added ten cases, stated that atopy is the base on which develop dermatoses from infancy to middle age; that, although immediate exciting factors vary, the fundamental predisposition is the same in all ages. He also stated that this tendency to sensitivity begins in embryonic life, continues through infancy and childhood and persists into adult life, tending to diminish in middle age.

The skin manifestations of atopic dermatitis are variable. In infancy the dermatitis is oozing, weeping and crusted; in childhood and young adult life it consists of slightly elevated papules and diffuse dry, scaly, hard and lichenified plaques. The distribution in cases of moderate involvement is in the flexural areas, primarily the antecubital and popliteal tissue, on the face, the forehead and the neck. The course is chronic, and most patients have severe exacerbations with intervals of months or years between the attacks.

Ætiology of Skin Manifestation and Cataract.

The ætiology of the skin manifestation and cataract is not settled, although numerous theories have been put forward and have been found wanting. Bellows (1944) summarizes the theories, which include metabolic, nutritional, hormonal and electrolytic imbalance. Other ætiological factors worthy of consideration are the following.

The Atopic or Allergic Theory.

It is believed by many (Cordes and Moreno, 1946; Beetham, 1940; Sulzberger, 1940; Sack, 1947) that the cutaneous lesion and the cataract are atopic phenomena. In addition to vascular structures there are three tissues in which the disturbed physiology of allergy may take place—namely, the skin, mucous membrane and smooth muscle. The crystalline lens may be the site of allergic reaction. The lens, like the skin, is ectodermal in origin, and it is possible that where the skin has been the seat of prolonged, extensive and severe hypersensitivity, an organ allied to it embryologically may ultimately come to participate in the hypersensitive process. This association constitutes the basis for describing this type of cataract as atopic (Sack, 1940).

Certain arguments against the allergic nature of the cataract require mentioning. (i) If disseminated neurodermatitis is so common, then why is the cataract so rare? From the cases reported, the average duration of the dermatitis before cataract develops is fifteen years. In most cases atopic dermatitis does not last so long as that, clearing either spontaneously or with medical assistance. (ii) Why is it that the cataracts are not always bilateral? It is not an invariable finding in allergy to have the disturbance universal in its distribution.

The Psychogenic Theory.

Most of these patients demonstrate nervous instability and exhaustion, and psychiatric maladjustment is frequent. They display general nervousness and irritability. In the case reported below an exacerbation of the neurodermatitis was always precipitated by emotional upset. Beetham (1940) concluded that the nervous state was due to the discomfort caused by the pruritic state and not an ætiological factor in its causation.

The Role of X-radiation.

It has been suggested that as many of these patients had been treated with X rays, therein lay the cause of the cataract. This factor is not now seriously considered; a large number of dermatological conditions are treated with X-radiation without the development of cataracts.

Embryological and Immunological Theories.

Daniel (1935) postulated the theory that the change in the function of the epithelial cells of the ciliary body, in the content of the aqueous fluid or in the level of permeability and selectivity of the lens capsule, can readily be reasoned to interfere with the efficient metabolism of the lens and its transparency, thereby producing a cataract in an otherwise normal eye. Actually, there is no proof of the changes so postulated. Beetham (1940) removed

aqueous from two of his patients and injected it intradermally, but failed to produce any reaction. Using lens material, Brunsting (1936) performed a skin test on one of his patients, also with negative response. One cannot at present postulate a change in capsular permeability to the contents of the aqueous as the immediate physical cause for the cataract formation in the entity under discussion (Sack, 1940).

Changes in the Lens.

Beetham (1940) describes two types of lenticular changes. The first is the typical and commonly seen complicated cataract. It usually begins in the posterior pole of the lens with increased iridescence, vacuoles and precipitates. Striation of the lens fibres is often present, and small punctate opacities are common. These changes spread peripherally, and similar changes are soon noted in the anterior cortex at the pole, immediately beneath the capsule. The changes progress until the entire cortex becomes cloudy to produce an intumescent or a homogeneous mature cataract.

The second type, that first described by Vogt (1922), is a dense irregular plaque of opacity in the pupillary area immediately beneath the capsule of the lens and possibly involving it to some extent.

Cowan and Klauder (1950) describe the lens changes after examining 100 patients with disseminated neurodermatitis, eight of whom had cataracts in some stage of development. The cataract nearly always appears first in the neighbourhood of the pole immediately in front of the posterior capsule. It begins with one or two transparent globules or flattened vacuoles, a few tiny white or yellowish opacities, and sometimes an iridescent crystal. These multiply and enlarge and coalesce until there is a mass of whitish, yellowish, transparent, translucent and opaque bodies of various shapes and sizes. It soon assumes a thin but dense, irregularly translucent, generally yellow disciform or rosette appearance, and lies just in front of and parallel to the posterior capsule. It grows forward and peripherally, but mostly toward the equator. At the same time or soon after, a process similar to that just described takes place in the anterior subcapsular zone of the lens. At some stage in the development of atopic cataract, a dense, bluish, pearly white, plaque-like opacity is sometimes found under the anterior capsule around the pole. Usually it is irregularly round, with scalloped or crenated edges and with extensions in the form of radiating solid or dotted lines towards the equator and the back of the lens. The capsule over this shield-shaped opacity may be wrinkled or elevated. The opacity itself is entirely cortical. Finally it reaches a stage which in all respects resembles the mature senile cataract.

The incidence of cataract in disseminated neurodermatitis is approximately 10%. Cowan and Klauder (1950) examined 100 consecutive patients with atopic dermatitis whose average age was 16·4 years. Of this group, eight patients were found to have cataract in some stage of development. The cataract was bilateral in five and unilateral in three. Beetham (1940) stated that 10% of all patients with disseminated neurodermatitis would develop cataracts. Only one case has been reported in the Australian literature (Tostevin, 1938).

From a survey of the literature, Sack (1947) concluded that the average age of onset of cataract was twenty-five years, and the average interval between the onset of the dermatitis and the cataract was fifteen years.

Results of Cataract Extraction in Disseminated Neurodermatitis.

Cordes and Moreno (1946) state that there are no special hazards in the removal of a cataract from these patients, except that intracapsular extraction should not be attempted, as the capsule is very thin. Sack (1947) states that the prognosis of cataract with neurodermatitis is good if the surgical treatment is skilfully effected.

Coles and Laval (1952) found that 63 cases of atopic cataract had been reported in the literature, and they found that in 36% the operative result was unfavourable. Of these, retinal detachment occurred in 14 or 22% of the eyes. Vogt (1922) referred to the extreme iridocyclitis which follows surgical procedures in this type of cataract. In the case reported by Coles and Laval (1952), severe iridocyclitis destroyed both eyes. Metzger (1932) referred to the frequent occurrence of retinal bleeding which followed cataract extraction. In view of these findings the operative prognosis must be regarded as bad, retinal detachment being the complication to be most feared.

It has been suggested that trauma may be the cause of retinal detachment. Balyeat (1937) considers retinal œdema to be the cause of the detachment, and Myleus (1944) favours vascular change in the retina as the cause. Coles and Laval (1952) are of the opinion that the primary cause of retinal detachment is a diseased vitreous, which does not hold the retina in place. While detachment may occur prior to surgery, it is more common afterwards.

Report of a Case.

Miss R. was first examined in August, 1951, when she was aged thirty-two years. The right eye had been very painful for several days, and the visual acuity in both eyes had been deteriorating since April, 1950. She had had disseminated neurodermatitis for many years. Her first dermatological episode occurred at the age of six years, when she had a rash which was stated to have been an allergy. When she was aged eleven years a rash commenced which was attributed to sensitivity to ivy. This persisted for several years. In 1943 she had conjunctivitis followed by a generalized rash, which commenced on the face and was accompanied by a feeling of heat throughout the body. This persisted for three to four months.

In 1944 she had another acute exacerbation, which coincided with the death of two of her brothers. The acute condition lasted approximately three months, leaving a chronic eczematous state.

In 1948 she had another acute exacerbation, in which the eczema was of a weeping type, and this she attributed to contact with paspalum. Since then she has had exacerbations and remissions, the exacerbations being precipitated by humidity and emotional upset.

At present the rash is confined to the flexures, the face and the neck, and when any exacerbation occurs the skin of the face shows the most pronounced changes. The skin of the flexures and arms is coarse and lichenified.

The only family history of allergic disorder is in a maternal aunt, who suffers from asthma.

Examination of the patient's eyes in August, 1951, revealed a large corneal ulcer in the right eye and a cataract in each eye. It was six weeks before the ulcer was completely healed and the eye white, and at that time the visual acuity was 6/60 in the right eye and 6/24 in the left eye; this improved to 6/36 and 6/18 respectively. Seven months later the visual acuity was less than 6/60 in the right eye and 6/36 in the left eye. In the right lens there was a large star-shaped plaque in the pupillary area, fundus details could not be made out, and there was a synechia at the 12 o'clock and 6 o'clock positions. In the left lens there was a small, round, calcified anterior subcapsular plaque, and emanating from it a diffuse opacity in the anterior cortex. The fundus was normal.

In October, 1952, a right combined extraction was performed. After the capsule had been broken, the plaque in the anterior cortex was expressed, but attempts to express the lens failed, so that a linear extraction had to be carried out. The post-operative course was uneventful, the patient being discharged from hospital on the eighth day. On February 12, 1953, the visual acuity with correction was 6/12, and she could read J2 type.

A fine posterior membrane was needled in June, 1953, after which the visual acuity with correction was 6/9 and she could read J1 type.

A combined extraction was performed because of previous iritis and because of the fear of post-operative iridocyclitis.

Summary.

1. Atopy is defined.

2. The ætiology of *neurodermatitis disseminata* (atopic dermatitis) and of the ocular complication — namely, cataract—is outlined.

3. The lens changes are described.

4. The results of cataract surgery in these patients are mentioned, and attention is drawn to the high percentage of complications following such surgery.

5. A case of *neurodermatitis disseminata* of long standing in which cataracts developed, with successful extraction of one cataract, is described.

References.

ANDREWS, G. C. (1946), "Diseases of the Skin", Third Edition, Saunders, 136.

BALYEAT, R. M. (1937), "Complete Retinal Detachment in Both Eyes in Neurodermatitis", *Am. J. Ophth.*, 20: 550; quoted by Coles and Laval (1952), *loco citato.*

BEETHAM, W. P. (1940), "Atopic Cataracts", *Arch. Ophth.*, 24: 21.

BELLOWS, J. C. (1944), "Cataract", Mosby, 470.

BRUNSTING, L. A. (1936), "Atopic Dermatitis of Young Adults; Analysis of Precipitating Factors in 101 Cases and Report of Ten Cases Associated with Juvenile Cataract", *Arch. Dermat. & Syph.*, 34: 936; quoted by Sack (1947), *loco citato.*

COCA, A. F. (1934), "Specific Diagnosis and Treatment of Allergic Diseases of the Skin; Present Status", *J.A.M.A.*, 103: 1275.

COLES, R. S., and LAVAL, J. (1952), "Retinal Detachments Occurring in Cataract Associated with Neurodermatitis", *Arch. Ophth.*, 48: 30.

CORDES, F. C., and MORENO, R. (1946), "Atopic Cataracts: Report of Four Cases", *Am. J. Ophth.*, 29: 402.

COWAN, A., and KLAUDER, J. (1950), "Frequency of Occurrence of Cataract in Atopic Dermatitis", *Arch. Ophth.*, 43: 759.

DANIEL, A. K. (1935), "Allergy and Cataracts", *J.A.M.A.*, 105: 481; quoted by Sack (1947), *loco citato.*

METZGER, E. (1932), *"Katarakt bei Neurodermatitis", Klin. Monatsbl. Augenh.*, 89: 821; quoted by Coles and Laval 1952), *loco citato.*

MYLIUS, K. (1949), *"Doppelseitige spontane Netzhautablösung bei 2 jugendlichen, seit Jahren an Neurodermatitis disseminata leidenden Patienten", Klin. Monatsbl. Augenh.*, 115: 247; quoted by Coles and Laval (1952), *loco citato.*

SACK, S. S. (1947), "Atopic Cataract. Report of a Case with Tabulated Summary of Previously Reported Cases", *Ann. Allergy*, 5: 353.

SULZBERGER, M. B. (1940), "Dermatologic Allergy", Thomas, 158.

TOSTEVIN, A. L. (1938), "Cataracta Dermatogenes or Cataract with Neurodermatitis Chronica", M. J. AUSTRALIA, 2: 647.

VOGT, A. (1922), *"Weitere Ergebnisse der Spaltlampenmikroskopie", Von Graefes Arch. Ophth.*, 109: 197; quoted by Beetham (1940), *loco citato.*

Reports of Cases.

A REPORT OF TWO CASES OF VOLVULUS OF THE SMALL INTESTINE.

By R. I. MITCHELL,

Assistant Surgical Registrar, The Royal North Shore Hospital of Sydney.

VOLVULUS of the small intestine is an uncommon condition giving rise to a characteristic clinical picture, causing a strangulating obstruction and requiring early intervention for its successful management.

Incidence.

In a review of 712 admissions for bowel obstruction at the Royal Victoria Hospital, Montreal (Ripstein and Miller, 1950) small intestine volvulus was found to be present in 23 cases (3·2%).

Thirty-six cases (6·9%) of small bowel volvulus were found in 520 patients with acute intestinal obstruction at the Massachusetts General Hospital (Sweet, 1935).

At the Royal North Shore Hospital of Sydney volvulus of the small intestine was found to be present in 11 of 150 patients submitted to operation for acute intestinal obstruction (7·3%).

Ætiology.

Volvulus of the small intestine may occur at any age. Its cause is usually ascertained at operation, but in some instances—the so-called idiopathic group—no reason for the volvulus is found.

Dott (1923) has described the gross developmental errors causing *volvulus neonatorum.* Congenital bands and congenital abnormalities in bowel rotation and fixation are frequently ætiological factors, and it is often surprising how late in life these may give rise to volvulus.

Peritoneal adhesions are the most common acquired lesion causing volvulus. Of 37 cases of small bowel volvulus at the Mayo Clinic (McKechnie and Priestley, 1936) 21 were caused by adhesions. A careful search is sometimes required to demonstrate a small band at the base of the mesentery, which approximates two points and acts as the axis about which the volvulus takes place. McWatters

FIGURE I.
Lipoma of the mesentery and ileum stretched over it (Case II).

(1945) believes that diet is an important factor in ætiology, stating that bulky farinaceous foods increase the incidence. Volvulus may also take place around tumours or in hernial sacs.

Case I.

P.P., a male child, aged thirteen months, was admitted to hospital on January 29, 1952, with a history of having fallen from his bed sixteen hours previously, and this had been followed eight hours later by vomiting, accompanied by screaming and restlessness. The infant's parents remarked that the child had become very pale with the onset of the vomiting.

Two months previously the child had had a similar attack of vomiting and screaming. An operation was performed elsewhere, at which an apparently normal appendix was removed through a gridiron incision.

Physical examination showed the patient to be a very pale child, listless, and showing no interest in his surroundings. No other abnormality was detected, and it was considered that his immediate condition was due to a head injury; the treatment advised was rest and observation.

The following morning the clinical picture was greatly changed. The child was vomiting frequently and effortlessly, his pulse rate was rising, and he had a distended abdomen. A rather vague mass was felt in the right iliac fossa, and bowel sounds were infrequent.

TABLE I.

Eleven Cases of Volvulus of the Small Intestine Found in 150 Admissions for Acute Intestinal Obstruction at The Royal North Shore Hospital of Sydney.

Case Number.	Sex.	Age. (Years.)	Site.	Ætiology.	Bowel Resection.	Result.
1	M.	61	Ileum.	Band.	Yes.	Died.
2	M.	52	Ileum.	No cause stated.	No.	Died.
3	F.	72	Jejunum and ileum.	Band.	No.	Recovered.
4	F.	43	Ileum.	No cause stated.	Yes.	Recovered.
5	M.	35	Jejunum and ileum.	(i) Short base of mesentery. (ii) Mobile caecum.	No.	Recovered.
6	M.	62	Ileum.	Malfixation of caecum and ascending colon.	No.	Recovered.
7	F.	58	Ileum.	Adhesions.	No.	Recovered.
8	M.	57	Ileum.	In inguinal hernial sac.	Yes.	Recovered.
9	M.	13 months.	Ileum.	Idiopathic.	Yes.	Recovered.
10	M.	36	Ileum.	Omental band.	No.	Recovered.
11	M.	2	Ileum.	(i) Lipoma of mesentery. (ii) Caecum and ascending colon mobile with dorsal mesentery.	Yes.	Died.

The child was submitted to laparotomy two hours later; in the interim intravenous therapy and continuous gastric aspiration had been instituted.

At operation, a volvulus of the terminal twelve inches of ileum was found to be present; the bowel was gangrenous and was therefore resected. Some of the mesenteric glands in this region were enlarged; but the caecum and ascending colon were well fixed, and no adhesion or congenital abnormality was present. An end-to-end anastomosis was carried out—the site of the anastomosis was within one inch of the ileo-caecal valve—and the abdomen was closed without drainage.

Post-operatively, gastric suction and intravenous therapy were continued and 1,000,000 units of penicillin administered daily.

On the third post-operative day the bowel sounds returned and the patient had a loose bowel action. The gastric tube was removed, and oral administration of fluids was commenced. Two days later intravenous therapy was suspended, as the child was then able to take sufficient fluids orally. The remainder of his convalescence was uneventful.

Case II.

K.S., aged two years, was a healthy male infant. He was admitted to hospital on January 20, 1953, with a history that he had commenced to vomit thirty hours previously, and had continued to do so until the time of admission. He had been given two enemas at home, which had yielded hard fæces and some mucus.

Physical examination showed the patient to be a pale child of the stated age lying listlessly in his cot. His abdomen was distended and tympanitic. A mass was present in the right iliac fossa, and the bowel sounds were accentuated.

A plain X-ray examination of the abdomen with the patient in the erect posture revealed multiple fluid levels in the left hypochondrium.

Intravenous therapy and continuous gastric suction were commenced, and the child was submitted to laparotomy.

At operation a large tumour was found in the mesentery of the ileum, around which a volvulus had occurred in a clockwise direction. The caecum and ascending colon were mobile, being attached to the posterior abdominal wall by a mesentery, but they were not involved in the volvulus. The tumour and segment of ileum tightly stretched over it were resected, and a lateral anastomosis was performed. The patient survived the laparotomy by twenty-four hours.

Autopsy revealed a recurrence of the volvulus. When the bowel was untwisted it was found that the caecum and ascending colon were very mobile, being attached to the posterior abdominal wall by a mesentery two to three inches in length.

Histologically the tumour was shown to be a lipoma.

Discussion.

The first case is an example of the idiopathic type of small intestine volvulus, whereas in the second two ætiological factors were present.

In both cases the striking clinical features were the great pallor, listlessness and prostration. Urgent hæmoglobin estimations were requested on each patient on admission to hospital, but the findings were within normal limits.

Clinical and radiological signs of acute intestinal obstruction were present, but it is the pallor and prostration, no doubt resulting from the vascular and neurogenic effects of torsion of the mesentery, which make volvulus give such a distinctive clinical picture, differentiating it from a simple non-strangulating obstruction. Other manifestations of strangulation, such as abdominal rigidity and increasing tenderness, are later to appear.

Conclusion.

Two cases of volvulus of the small intestine in infants have been encountered within a period of one year at the Royal North Shore Hospital. A review of 150 cases of acute intestinal obstruction at this hospital showed eleven to be caused by small bowel volvulus.

Acknowledgements.

I wish to thank the General Medical Superintendent of the Royal North Shore Hospital of Sydney, Dr. Wallace Freeborn, for permission to publish the case reports. I am indebted to Dr. L. S. Loewenthal and Dr. T. F. Rose, under whose care these patients were admitted to hospital, for their advice in the preparation of this paper.

References.

DOTT, N. M. (1923), "Anomalies of Intestinal Rotation: Their Embryology and Surgical Aspects: Report of Five Cases", *Brit. J. Surg.*, 2: 251.

MCKECHNIE, R. E., and PRIESTLEY, J. T. (1936), "Volvulus of the Small Intestine; Report of 37 Cases", *Am. J. Surg.*, 34: 286.

MCWATTERS, R. C. (1945), "Volvulus of the Small Intestine", *Brit. M. J.*, 2: 626.

RIPSTEIN, CHARLES B., and MILLER, G. GAVIN (1950), "Volvulus of the Small Intestine", *Surgery*, 27: 506.

SWEET, R. H. (1935), "Volvulus of the Caecum: Acute and Chronic", *New England J. Med.*, 287: 213.

A CASE OF PROBABLE OVARIAN AGENESIS WITH TURNER'S SYNDROME IN A FEMALE CHILD AGED SEVEN MONTHS.

By ELIZABETH K. TURNER, M.D.,
Melbourne.

IN 1938 H. H. Turner described the triad of infantilism, *pterygium colli* or webbed neck and *cubitus valgus*. This was the first record of this syndrome appearing in English literature, although Funke in 1902 gave the first account of deficient sexual development combined with *pterygium colli*, and such cases are recorded in the French and German literature prior to 1938.

In 1942, Varney, Kenyon and Koch, and Albright, Smith and Fraser showed by hormone analysis of the urine that these cases of deficient sexual development were due to a primary deficiency of the ovaries.

Since 1942 at least 60 cases have been published, and probably well over 100 cases have been diagnosed at various clinics.

The youngest recorded patients have been infants aged about three years. Wilkins states that "the syndrome presents one of the most striking and clear-cut clinical pictures encountered in endocrine practice".

The features are stunting of growth, the patients usually attaining an eventual height of 50 to 58 inches, and infantile breasts, labia, clitoris, vagina and uterus, as well as rudimentary or missing ovaries. Sexual hair is usually present, but is scanty and often does not appear until

FIGURE I.

Patient, aged fourteen months, showing stunted stature.

the age of fourteen or sixteen years. *Maturation of bones* is approximately normal, as also is the development of *the teeth.* Urinary gonadotropin (follicle-stimulating hormone from the pituitary) is greatly increased after the age of twelve years, and the excretion of 17-ketosteroids is *normal or slightly reduced.* Added to this there are a number of varied congenital anomalies, the most striking of which is webbed neck, often associated with coarctation *of the aorta, abnormalities of the digits, lymphangiectatic* œdema, cystic bony changes, azygos lobe of the lung, *anomalies of the renal pelvis et cetera.*

The mandible and chin are frequently poorly developed, so that the features of many patients present a striking *similarity; in fact, the characteristic physiognomy is* described as "bec de lièvre" or hare beak.

Lissen et alii (1947) describe 25 cases and make the following statement:

> We are of the opinion that a typical case of genital infantilism, short stature, stocky build with shield chest, webbed neck and cubitus valgus constitutes a syndrome so unique that the assumption of congenital ovarian aplasia seems justified, even without corroborative high gonadotropin titres or pathological verification.

One-third to one-half of all patients with hypogonadism present pterygium; in fully one-third of recorded cases of pterygium syndrome primary hypogonadism is present; two-thirds of the patients with pterygium also have coarctation of the aorta. These abnormalities represent manifestations of the same syndrome of defective development and malformations. In both ovarian agenesis and the pterygium syndrome the same ætiological factor has evidently operated at the organo-genetic stage of embryonic development.

Clinical Record.

A girl, J.G., was first examined at the age of five and a half months. She was the first child of normal parents, and there are no familial illnesses or abnormalities in the

FIGURE II.

Showing lymphangiectatic œdema of lower extremities.

family. During the pregnancy her mother suffered no *infections or hæmorrhages,* and was well except for hypertension in the third trimester, for the treatment of which her diet was somewhat restricted.

The infant was born at *full term; the labour was difficult,* the cord being round the baby's neck, and there was *some difficulty in resuscitation.* The birth weight was four pounds eight ounces, and the length was 19 inches at one month. The infant was not jaundiced and sucked well, *being breast fed until the age of three months,* and then weaned on to a cow's milk mixture. Since the age of three months the infant had been subject to frequent attacks of *screaming, accompanied by pyrexia and scalding of the* urine. Her doctor had discovered numerous pus cells in her urine on several occasions, and a strain of *Bacterium coli* had been grown on culture.

When the infant was aged five months, 16 cubic centimetres of *"Uriodone" were injected intramuscularly (eight* cubic centimetres into each buttock), with 300 units of a *hyaluronidase preparation.* A skiagram showed that both renal shadows were of normal size, position and outline. No opaque calculus was evident in the kidneys,

ureter or bladder. The dye was excreted through both kidneys in normal time. The upper poles of both kidneys were drained through small pelvocalyceal systems whose small upper ureters (really elongated communicating channels) ran into the main ureters at the normal pelvi-ureteral junction. There was no hydronephrosis on either side, and the bladder shadow was large; it was noted that the bladder did not empty completely on micturition.

At this stage the infant attended the Melbourne Children's Hospital out-patient department. She was an undersized baby, weighing eleven pounds. She had a peculiarly mature face with an alert expression, an underdeveloped mandible and soft-tissue webbing of the neck. The heart was enlarged and a soft systolic bruit was audible all over the precordium. Her complexion was pale with a slight cyanotic tinge in the circumoral region, and she had

FIGURE III.
Showing soft-tissue webbing of neck and typical facies.

pitting œdema of both feet and legs. She also had peculiarly shaped ears and hypertrophy of the fourth digit on the left hand (see Figures I, II, III and IV).

The hair of the head was fair, sparse and of normal texture, with a lowered hair line on the neck. The thorax was well developed and the carrying angle of the elbow was 160°.

No abnormality was detected in the abdomen and external genitalia.

A blood examination revealed a hæmoglobin value of 70%, or 10·2 grammes *per centum*. The red blood cells numbered 3,800,000 per cubic millimetre; the white blood cells numbered 6900 per cubic millimetre, the differential count being normal for the infant's age; platelets were plentiful.

Electrocardiographic examination failed to reveal any axis deviation, but right ventricular hypertrophy was detected in the chest leads, with evidence of a possible early right anterior branch block.

Fluoroscopic screening of the chest disclosed that the cardiac contour was not suggestive of pure coarctation. There was right auricular enlargement and the apex was tip-tilted, suggesting an enlarged right ventricle. The lung fields appeared clear. There was no detectable hypertrophy of collateral thoracic or pectoral vessels.

An X-ray examination of the long bones revealed no abnormality except slight cupping of the wrists and knees.

The radial pulse was very strong, and the blood pressure recorded in the upper extremity was 146 millimetres of mercury, systolic, and 85 to 90 millimetres, diastolic.

The femoral pulses were scarcely palpable, and the blood pressure in the lower extremity was not recordable. The mother said that the child was hot and clammy in the upper extremities and chest, but cold in the lower extremities and the lower half of the body.

The Mantoux test produced no reaction.

FIGURE IV.
Showing low hair line and pterygium colli.

Laboratory Findings.

A catheter specimen of the urine contained 90 leucocytes per high power field on one occasion, and on culture yielded a growth of *Bacterium coli* and non-hæmolytic streptococci.

The details of blood chemistry estimated when the child was fourteen months old are as follows. The hæmoglobin value was 60%. The hæmatocrit reading was 30 millimetres. The sodium content of the blood was 345 milligrammes per 100 cubic centimetres (140 milliequivalents per litre); the potassium content was 19·5 milligrammes per 100 cubic centimetres (5 milliequivalents per litre); the calcium content was 11·5 milligrammes per 100 cubic centimetres (5·8 milliequivalents per litre); the total sodium, potassium and calcium content was 151 milliequivalents per litre. The alkali reserve was 49·2 volumes of carbon dioxide per 100 cubic centimetres (20·4 milliequivalents per litre); the chloride content was 640·0 milli-

grammes per 100 cubic centimetres (109·4 milliequivalents per litre); the phosphate content was 4·2 milligrammes per 100 cubic centimetres (2·4 milliequivalents per litre); the total protein content was 6·37 grammes per 100 cubic centimetres (15·5 milliequivalents per litre); thus the alkali reserve, the chloride and phosphate contents and the total protein content of the serum equalled 148 milli-equivalents per litre.

Hormone analysis of the urine was not assayed because an elevation of the follicle-stimulating hormone is not found before the age of ten to twelve years, when the secretion of the pituitary gonadotropin first begins.

The infant also has an allergy to cow's milk, but apart from dietetic instruction and antibiotic therapy for attacks of urinary infection, no substitution therapy with gonadal hormones has been given.

Discussion.

According to Wilkins (1950), substitution treatment with gonad hormones is rational; 0·5 to 5·0 milligrammes (usually one milligramme) of stilbœstrol is given by mouth for several months. When the genitals have reached the stage of adolescent development, or endometrial bleeding occurs, cyclic treatment is instituted. For two weeks stilbœstrol is given alone; then for one week stilbœstrol and progesterone are given (10 to 30 milligrammes per day), then no drugs are given for the fourth week. Bleeding usually occurs two or three days after the treatment is stopped.

If sexual hair is also deficient testosterone (25 milligrammes) is given intramuscularly twice a week for the first three weeks of the cycle; however, this may cause enlargement of the clitoris and huskiness of the voice.

Œstrogen stimulates development of the breasts, the labia minora, the vagina and the uterus; progesterone stimulates development of the mammary glands; growth, however, seems permanently stunted, and of course fertility is not attained.

At laparotomy or by peritoneoscopy the uterus and Fallopian tubes are found in an infantile state; the ovaries are represented by two thin whitish bands running parallel to the Fallopian tubes in the broad ligament. These are composed of no active ovarian germ cells or epithelial cells, and are mainly cells of the ovarian stroma type.

During embryological development it may be that some noxious influence causes an arrest of growth in the indifferent stage of the genital gland. The kidneys and genital glands develop very close to each other, the genital ridge arising as an area of thickened epithelium on the medial side of the mesonephric (Wolffian) ridge; cellular cords appear, which are called the medullary cords of the ovary, and the sex cells which they contain proliferate and develop into oocytes which undergo degeneration and disappear.

A second invasion of gonocytes now grows into the mesenchyme from the yolk sac and margins of the embryonic area; these cells form the ova. It would appear that arrest in development may take place before this second invasion of sex cells, so that the ovary does not develop beyond the primary medullary ridge stage; the same arrest may also be responsible for the renal anomalies found, and perhaps for the several cases of adrenal cortical hypoplasia discovered in such subjects *post mortem*. This hypoplasia may determine the observed diminution in the 17-ketosteroid excretion.

There is a striking preponderance of females reported with this condition; indeed, records of three males only appear. This is important, as persons who lack gonads apparently have entirely normal female differentiation of the genital ducts and other sex organs.

Jost (quoted by Wilkins, 1950) has surgically castrated rabbit embryos before sex differentiation has occurred and all these embryos when born at full term have developed as females. This suggests that sex determination is not entirely dependent on chromosomal pattern, and unless testes are present to antagonize the autonomous feminizing tendency (which may be under the influence of maternal œstrogen), all the embryos will develop as females.

Theories to explain the stunting of growth are numerous. It is believed that œstrogen may have a slight direct effect on the rate of growth in the epiphyseal cartilage, œstrin is known to cause proliferation of many tissues which after the menopause show a propensity for atrophy—for example, osteoporosis and atrophy of the skin—and in granulosa cell tumour growth in height may be increased; on the other hand, castration before puberty may lead to increased growth and eunuchoid proportions.

Primary ovarian deficiency leads to secondary changes in the anterior lobe of the pituitary, which in turn may interfere with production of the growth hormone by the pituitary. Autopsy findings summarized by Pich (1937) showed minor changes in the histological picture of the anterior pituitary lobes of some subjects, with an increase in chromophobe cells.

Against this hypothesis is the fact that castration in males does not lead to decreased stature, in spite of the fact that over-production of follicle-stimulating hormone takes place in the pituitary.

Tumours of the adrenal cortex cause pronounced sexual and somatic precocity, as the adrenal cortex produces steroids very similar to testosterone, which is known to be an important growth hormone. In the condition under discussion the 17-ketosteroids remain at the prepubertal level, which suggests decreased adrenal cortical activity.

In this condition there is no evidence of hypofunction of the hypophysis or thyroid gland, and the cardiac and renal anomalies are not *per se* sufficient to produce such stunting.

The dwarfism produced is generally regarded as being a "genetic defect in development which happens to be 'linked' fairly regularly with other genetic defects to constitute a congenital syndrome" which arises as the result of some widespread arrest during the early stages of fœtal development.

Summary.

A diagnosis of ovarian agenesis with Turner's syndrome has been made empirically in the case of an infant, aged seven months, who shows features typical of this clear-cut endocrine picture. This infant presents dwarfism, pterygium colli, a variant of coarctation of the aorta, digital and renal anomalies, and the typical facies.

Hormonal analysis of the urine has not been undertaken, as the typical elevation of the follicle-stimulating hormone does not occur before the age of ten or twelve years. For humane reasons laparotomy has not been performed to confirm the diagnosis, but it is the writer's intention to keep this infant under surveillance for many years.

A discussion of the theories of causation of dwarfism in this condition is given, and some interesting facts about sex differentiation emerge.

References.

ALBRIGHT, F., SMITH, P. H., and FRASER, R. (1942), "A Syndrome Characterized by Primary Ovarian Insufficiency and Decreased Stature", *Am. J. M. Sc.*, 204: 625.

FLAVELL, G. (1943), "Webbing of the Neck with Turner's Syndrome in the Male", *Brit. J. Surg.*, 31: 150.

FUNKE (1902), quoted by Flavell, G., *loco citato*.

LISSEN, H., CURTIS, L., ESCAMILLA, R., and GOLDBERG, M. (1947), "The Syndrome of Congenitally Aplastic Ovaries with Sexual Infantilism, High Urinary Gonadotropins, Short Stature, and Other Congenital Abnormalities", *J. Clin. Endocrinol.*, 7: 665.

PICH, G. (1937-1938), quoted by Albright, F., Smith, P., Fraser, R. (1942), "A Syndrome Characterized by Primary Ovarian Insufficiency and Decreased Stature", *Am. J. M. Sc.*, 204: 625.

TURNER, H. H. (1938), "Syndrome of Infantilism, Congenital Webbed Neck, and Cubitus Valgus", *Endocrinology*, 23: 566.

WILKINS, L. (1950), "The Diagnosis and Treatment of Endocrine Disorders in Childhood and Adolescence".

VARNEY, R. F., KENYON, A. T., and KOCH, F. C. (1942), "Association of Short Stature, Retarded Sexual Development and High Urinary Gonadotropin Titres in Women with Ovarian Dwarfism", *J. Clin. Endocrinol.*, 2: 137.

Reviews.

Incompatibility in Prescriptions and How to Avoid It: With a Dictionary of Incompatibles. By Thomas Stephenson, D.Sc., Ph.C., F.R.S.E. Sixth Edition, by James Burnet, M.A., LL.B. (London), M.D., F.R.C.P.E.: 1953. Edinburgh: "The Prescriber" Publishers, Limited. 8¼" × 5¼", pp. 70.

THIS book provides practical information on the question of incompatibility in prescriptions presented in a straightforward manner which makes for easy consultation. The basic definition of the book is that "a prescription is said to be incompatible when the ingredients are opposed in character or interact one with another so as to interfere with the elegance, efficacy or safety of the resulting medicament". The first part of the book describes the general principles of incompatibility under the three main headings of chemical incompatibility, physical or pharmaceutical incompatibility, and therapeutic incompatibility. The second part consists of a dictionary of incompatibilities in which is provided an alphabetical list of drugs with their doses, solubilities and incompatibilities. As a final aid to easy reference, an index, in which the drugs are listed by their English names, provides many cross-references which would otherwise be missing. The days of the complex prescription are largely over, but the opportunity for prescribing incompatibilities still remains, and those who are not skilled in this matter would be well advised to have some such reference book as this at hand.

Dermatologic Formulary: From the New York Skin and Cancer Unit, Service of Dermatology. Edited by Frances Pascher, M.D.; 1953. New York: Paul B. Hoeber, Incorporated. 7¼" × 5", pp. 160. Price: $3.00.

THIS book is designed to cater for the needs of the dermatologist and possibly the general practitioner, and lists the most proven and useful dermatological prescriptions which have been well tried by leading dermatologists over the years. A good feature, not produced in most hospital pharmacopœias, is that the prescriptions are accompanied by short and simple explanations of their uses, indications and contraindications, the latter being especially useful. In many instances instructions regarding the mixing of ingredients are also given.

A Ciba Foundation Symposium: The Spinal Cord. Consulting editors, J. L. Malcolm, M.B., Ch.B., B.Med.Sc., and J. A. B. Gray, M.A., M.B., B.Chir., G. E. W. Wolstenholme, O.B.E., M.A., B.M., B.Ch., assisted by Jessie S. Freeman, M.B., B.S., D.P.H.; 1953. London: J. and A. Churchill, Limited. 8" × 5½", pp. 312, with 112 illustrations. Price: 30s.

THE latest volume of the Ciba Foundation Symposia deals with the anatomy, physiology and pharmacology of the spinal cord. In twenty papers, each with discussion, leading authorities from nine nations outline the present position regarding the constitution and functional activity of this important section of the central nervous system. The papers are of very high standard, often controversial and always difficult to follow by anyone who is not working in the higher reaches of this branch of studies. The subjects discussed include synaptic transmission at the motoneuron, the physiological function of the monosynaptic reflex, the organization of the monosynaptic and disynaptic pathways, the relation of acetylcholine to nerve transmission and the effect of anticholinesterases on the spinal cord, the effects of various other drugs on the spinal cord and nerve conduction in poliomyelitis. The discussions following the papers were particularly lively and show much difference of opinion on important points.

The neurophysiologist will learn much and get much stimulation, if not in the spinal cord, certainly in the higher brain, from reading this book. Some clinicians may learn much from the papers, but a considerable knowledge of neurology is necessary.

Trends of Life. By F. Wood Jones, F.R.S.; 1953. London: Edward Arnold and Company. 7½" × 5", pp. 192. Price: 10s. 6d.

PROFESSOR FREDERICK WOOD JONES does not mind being branded as a biological heretic—indeed he seems rather to enjoy provoking the wrath of the orthodox. In this little volume he states in simple terms what may be regarded as his *credo* in regard to life and living things. Life, he suggests, is something that is *sui generis*: "there is a great gulf fixed between the living and the non-living". He discusses the concepts of vitalism and of teleology and confesses adherence to both for reasons based purely on the findings of natural science. He defends Lamarckianism and develops in some detail his well-known opposition to orthodox Darwinism. All this he does with a wealth of illustration drawn from a lifetime of observation of living things, both in the field and in the laboratory, and he caps it with a realistic assessment of modern man that leaves little room for rosy optimism about the evolution of supermen. Even those who disagree with his conclusions will appreciate the lucidity of Professor Wood Jones's presentation and the easy charm of his literary style. His rebellion against orthodoxy will be approved by all except those who do not appreciate the fact that dogma is an anomaly in the scientific world.

CIBA Foundation Colloquia on Endocrinology. Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., assisted by Jessie S. Freeman, M.B., B.S., D.P.H.; 1953. Volume VI. Hormonal Factors in Carbohydrate Metabolism: London: J. and A. Churchill, Limited. 8" × 5¼", pp. 364, with 78 illustrations. Price: 35s.

THIS volume of the CIBA Foundation Colloquia on Endocrinology has a series of papers with discussions on the metabolism of carbohydrates and fats and the effects on the various enzyme systems concerned of various hormones. The authors of the papers are authorities on the aspects treated from Europe and America, with Professor C. H. Best, of Toronto, as the chairman.

The papers are divided into six groups. Part I deals with enzyme systems concerned in carbohydrate metabolism and there are three papers on less well-known aspects of the subject including the enzymic synthesis and structure of glycogen. Part II deals with hormonal control of the interconversion of carbohydrate, protein and fat and there are five papers. Part III deals with the influence of the adrenal cortex on carbohydrate metabolism and there are four papers including one on clinical observations on metabolism in obesity. Part IV consists of one paper on hormonal control of glycogen storage. Part V deals with the influence of insulin on carbohydrate metabolism and there are six papers including a paper by C. H. Best on the influence of insulin on carbohydrate metabolism and one by R. D. Lawrence on human and experimental diabetes. Part VI deals with sex hormones, pregnancy and carbohydrate metabolism, and there are five papers including one by J. P. Hoet, of Louvain, on pregnancy and diabetes.

The papers are all of very high standard and the discussions very interesting, particularly where the clinicians try to get the laboratory experts to clarify certain points. There is much new or not yet well-known material in the papers, and anyone interested in carbohydrate metabolism and diabetes, whether laboratory worker or physician, will find much of interest and value in this volume. It covers a very wide field and does it very thoroughly.

Pathology of Tumours. By R. A. Willis, D.Sc., M.D., F.R.C.P.; Second Edition; 1953. London: Butterworth and Company (Publishers), Limited. Sydney: Butterworth and Company (Australia), Limited. 10" × 7", pp. 1066, with 500 illustrations. Price: £5 14s.

The advent of a second edition of this text by R. A. Willis will be welcomed by all interested in this subject. The first edition was very well received and has on close acquaintance during the past five years proved its usefulness to pathologists and surgeons. The new edition has almost exactly the same number of pages and illustrations, and there are only minor alterations in the text.

Willis's definition of a tumour is an abnormal mass of tissue, the growth of which exceeds and is uncoordinated with that of the normal tissues and persists in the same excessive manner after cessation of the stimuli which evoked the change. Nicholson's "failure of co-ordination" or Needham's "escape from the controlling pattern or individuation field" is the essential quality in a neoplasm. Willis's criticism of the virus theory has gained weight since the first edition, owing to the work of Passey and others. Filterable agents have not yet been demonstrated in malignant mammalian tumours. The view that the so-called tumour "viruses" may not be parasitic organisms at all but abnormal proteins of the cells themselves is gaining increased support.

Willis is still somewhat dubious as to a true increase in lung cancer. He points out that though there has been a ninefold increase in certified deaths from lung cancer in England and Wales in 1947 as compared with 1928, yet this great increase is not reflected in the autopsy records of many large hospitals, which show less than a twofold increase. Is this, he asks, an increased recognition of

thoracic cancer due to other factors, for example, greater surgical interest in the disease, greater competence of pathologists *et cetera*?

In the first edition he found that the evidence afforded strong grounds for suspecting the carcinogenic results of smoking, but now he states it is wellnigh proved that smoking is an important cause of lung cancer, though elucidation of many points is still needed.

He believes that the diagnostic reliability of sputum examination for cancer cells is over-estimated. The danger of false positive reports even by experts outweighs the value of the method.

Cancer research organizations and cancer cause and cure propagandists might ponder on Willis's statement that no epoch-making discovery in tumour pathology has been made in the last five years, though our knowledge has been amplified and clarified in many directions.

Those interested will find some interesting revisions of special tumour pathology—for example, of myoblastomata, tumours of chemo-receptors, Brenner and masculinizing tumours of the ovary, and the histogenesis of pigmented moles and melanomata.

On all subjects Willis gives his personal views and draws from his large experience, and this and a talent for lucid writing and inspiring interest are the virtues of this great work. He may not always be proved right in the long run, but errors will probably be less than in any comprehensive book of this type.

William Cheselden, 1688-1752. By Zachary Cope, K.B., M.D., M.S., F.R.C.S.; 1953. Edinburgh and London: E. and S. Livingstone, Limited. 9" × 6¼", pp. 120, with 24 illustrations. Price: 20s.

YET another handsome biographical masterpiece bearing on the history of surgery comes to us from the enterprising firm of the Livingstones in Edinburgh. Their latest product is a happy combination of fine printing, worthy subject matter, and sound presentation; and this time we are treated to an excellent exposition of the life and work of William Cheselden. It cannot be stated unequivocally that Sir Zachary Cope has used his literary gifts to the fullest extent in the writing of this book, but it is obvious that the research has been thorough, and the reader is left with a clear impression of the character of Cheselden and of his many original contributions to surgical education and practice.

There can be little doubt that the founder of modern surgery possessed certain valuable traits which caused him to become a perfectionist in the art and science of surgery, to preserve a fearless integrity in all public or professional dealings, and at all costs to bring about a radical improvement in the standards of surgical education throughout Great Britain. Furthermore, it stands to his everlasting credit that the motive of financial gain was of secondary importance in the fulfilment of his surgical ambitions, and in all his deep-laid schemes to raise the craftsmen of his specialty in status and respectability. He was indeed the first of an illustrious dynasty of scientific surgeons which can be traced in a direct line through John Hunter and Astley Cooper to the brilliant company of surgeons who carried their best traditions far on into the nineteenth century.

After serving the long apprenticeship of seven years required of an aspiring hospital surgeon, Cheselden passed his qualifying examination at the old Barber-Surgeons' Hall in Monkwell Street, London, and at the age of twenty-three he settled down to wait patiently for private practice, and for an appointment to the surgical staff of Saint Thomas's Hospital, which did not come for another seven years. In the long interval he established the first regular and systematic course for the thorough teaching of anatomy ever held in London, employed the young Francis Hauksbee, F.R.S., to expound the basic sciences to his pupils, printed a detailed syllabus of the lectures, and in 1713 published "The Anatomy of the Humane Body", which maintained its popularity with the profession for nearly a century.

There are interesting chapters dealing with the work of Cheselden at Saint Thomas's Hospital, the Westminster Infirmary, Saint George's Hospital and, lastly, at the Royal Hospital, Chelsea, to which place he mysteriously retired in 1737, almost certainly with the one purpose in view—to devote all his energies to the cleverly organized task of tactfully bringing about a permanent separation of the surgeons from the barbers, a delicate operation which was smoothly accomplished in May, 1745.

Many surgeons, ophthalmologists and medical historians will appreciate the wealth of material compressed into the illuminating accounts of Cheselden's operations on the eye, and of the determined efforts made by the great surgeon and his colleagues, James and John Douglas, to explore the most advantageous routes for approaching the bladder in order to "cut for the stone". James Douglas (of the "Pouch" and "Fold") was responsible for the superb anatomical preparations which served a useful purpose in devising a new technique for the suprapubic and perineal operations of lithotomy with a minimum of injury to vital structures in the path of the knife.

This small book is a distinct contribution to the history of surgery, has a number of splendid illustrations, and is worthy of a place in all medical libraries.

Notes on Books, Current Journals and New Appliances.

Australasian Annals of Medicine, Volume II, Number 2.

THE issue of *Australasian Annals of Medicine* for November, 1953 (number 2 of Volume II), maintains the high standard set from its first issue by this young journal. The production is attractive and excellent in every way. The contributed articles are a good sample of Australian medicine, but we are sorry to note that New Zealand is not represented. E. G. Saint, W. E. King, R. A. Joske and E. S. Finckh examine the course of infectious hepatitis on the basis of 123 cases. F. F. Rundle, L. R. Finlay-Jones and K. B. Noad present a quantitative analysis of the orbital tissues from a patient with malignant exophthalmos. T. E. Lowe discusses the intake and output of water in the control of body water content. J. L. Allsop reviews the literature on thallium poisoning and presents details of 18 new cases. I. S. Collins gives an account of an hæmorrhagic syndrome due to a circulating anticoagulant. H. N. Robson presents and discusses a series of 17 cases of myeloscleroses. F. Avery Jones and W. E. King describe a study of acute gastric ulcers causing hæmorrhage, on the basis of 1223 admissions to hospital for hæmatemesis and melæna; the paper comes from Central Middlesex Hospital, London, where the Australian author (King) has been working during tenure of a Nuffield Dominion Fellowship. H. D. Breidahl, with the technical assistance of Jeanne Upfill, presents a clinical and biochemical study of the use of cation exchange resins in œdema. R. M. Mills discusses tuberculous infection in infants under the age of two years. C. R. B. Blackburn, A. E. McGuinness and I. Kaldor describe and emphasize the value of the removal of excess body iron in hæmochromatosis by repeated venesection. I. Kaldor discusses the value of serum iron and iron-binding capacity measurements in clinical diagnosis; this is the third of a series of studies on intermediary iron metabolism.

Australasian Annals of Medicine has now been in existence for two years and, having established itself quietly as a journal of high quality, is now about to branch out. In 1954 it is to be published quarterly in February, May, August and November. The subscription rate will be three pounds *per annum*, payable in advance to The Royal Australasian College of Physicians, 145 Macquarie Street, Sydney.

Family Doctor. Published monthly by the proprietors, the British Medical Association, Tavistock Square, London, W.C.1. Sole agents for Australia and New Zealand: Gordon and Gotch (Australia), Limited. Subscription for twelve months: 20s. (sterling), including postage.

AN important newcomer to "Family Doctor" in the December issue is Dr. Walter C. Alvarez, of the Mayo Clinic; in his well-known wise and happy style he writes an article on "How to Live with Your Nerves", the first of a series of articles. In "Baby Discovers its Parents" the camera is used with rare charm to show infant experiences. Indeed, the camera contributes notably to the whole issue. Sir Russell Brain writes on the Royal College of Physicians of London. Other articles deal with the facts about bread, middle-aged knees, grey hair in a child, having an operation, the Bates myth about eyesight, frozen foods and so on. The general standard of the material and its presentation are as high as ever. *Family Doctor*, as a magazine on medical matters for lay people, can be commended without reservation. A free sample copy will be sent to any doctor who applies to the Editor of THE MEDICAL JOURNAL OF AUSTRALIA.

The Medical Journal of Australia

SATURDAY, JANUARY 9, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

ATHEROSCLEROSIS.

ONE of the most controversial subjects in present-day medicine is the relationship between, on the one hand, blood and dietary cholesterol contents and, on the other, the incidence of atherosclerosis and degenerative diseases of the heart associated with it. Degenerative diseases of the heart—coronary heart disease, *angina pectoris*, chronic myocarditis and myocardial degeneration, are taking an increasing toll of lives all over the world, but particularly in the wealthier countries and notably amongst people in the age group fifty to sixty-five years. Ancel Keys[1] has discussed the statistical aspects and the possible causation of these diseases in a thoroughly documented paper entitled "Atherosclerosis: A Problem in Newer Public Health".

Taking as his basis the total death rates from all causes in the United States in 1949 for each age group and sex, expressed as 100, Keys gives the corresponding figures for the period 1947-1949 for sixteen other countries, mostly countries in which life is fairly easy. In the age groups forty to forty-four years, fifty to fifty-four years and sixty to sixty-four years, in only a few cases are the figures over 100. Thus, for Australia, in the fifty to fifty-four years group they are for males 87 and for females 96; in the sixty to sixty-four years group they are 94 for males and 94 for females. In contrast, the figures for Ireland are in the fifty to fifty-four years group 57 for males and 87 for females, and in the sixty to sixty-four years group 69 for males and 88 for females. For Norway, the figures in the fifty to fifty-four years group are 53 for males and 65 for females, and in the sixty to sixty-four years group 54 for males and 68 for females. The figures for Portugal are very close to those for the United States; but when the deaths from all diseases of the circulatory system are separated and compared, the results are striking. These are in the fifty to fifty-four years group for men in Portugal 1·8 per thousand deaths and in the United States 5·9; in the sixty to sixty-four years group they are 5·2 per thousand for Portugal and 13·9 for the United States. For the other countries, too, the differences in the figures compared to those for the United States in total age-specific death rates could be largely or wholly explained by differences in the death rates from degenerative heart disease. These results suggest that some differences in the mode of life of people in the different countries are responsible for the varying incidence of degenerative heart diseases. A belief which is very widely held is that atherosclerosis and conditions related to it are associated with a high blood cholesterol content, which is due directly to a high intake of cholesterol in the diet, particularly in such foods as milk, milk products and eggs. Expressions of opinion can be found in the literature all the way from a denial that cholesterol, whether in the blood or in the food, has anything to do with the matter, to such statements as the following, made by William Dock[1] in a recent editorial in *The Journal of Clinical Nutrition*: "Since two centuries of practical and experimental study have failed to convince some physicians that diets of salt pork, beans and flour may be inadequate for maintaining health, it probably will take a long time to convince the profession that diets rich in eggs, butter and cream cause the disease which now kills nearly one out of every three physicians. The biologist knows that such diets are as alien to adult mammals as those which cause scurvy, beriberi and pellagra." One wonders who is the author's "biologist".

What are the facts of the relation between cholesterol and other lipoids and the degenerative diseases of the heart? Keys points out that, compared with healthy persons of the same age, patients who have definite *angina pectoris* or who have survived myocardial infarction tend to have blood serum characterized by high cholesterol and certain lipoprotein concentrations, a high cholesterol-phospholipid ratio and a larger proportion of cholesterol in the β lipoprotein fraction; but they do not necessarily have all or any of these. Persons with diabetes, myxœdema and nephrosis tend to have a similar blood picture, and among these patients there is a high incidence of atherosclerosis and degenerative heart disease. In animal experiments, diets of high cholesterol content produce high blood cholesterol levels and atherosclerosis. Further, there are abnormal amounts of cholesterol in the walls of atherosclerotic arteries. How reliable are these facts in individual cases? Whenever groups of individuals are compared, all the measurements seem to show statistically significant correlations between the measurements and the presence or absence of the tendency towards degenerative heart disease. But when one comes to consider individuals, the correlations are of a very low order, whatever measurement is taken—total cholesterol content, cholesterol/phospholipid ratio, "giant molecules" of the S_f 10-20 and S_f 12-20 varieties, β lipoproteins and so on. Keys has shown by careful statistical analysis of his own and other observers' figures that there is no evidence for an important difference between S_f 10-20 lipoprotein and cholesterol measurements as devices to differentiate between clinically healthy people and patients with definite coronary disease, and that neither measure is acceptably reliable in dealing with individuals. While it is true that an apparently

[1] *J. Mt. Sinai Hosp.*, July-August, 1953.

[2] *J. Clin. Nutrition*, March-April, 1953.

normal individual may show a very high blood cholesterol level within normal limits, there is no doubt that high serum levels of cholesterol are undesirable. What is the value of the evidence that high cholesterol intake in the food is harmful? All animals, including man, synthesize cholesterol, mainly in the liver, and eliminate it in the bile and by chemical degradation. When exogenous cholesterol is supplied, the synthesis in the liver is depressed. Some animals, such as the rabbit and the chicken, consume little or no cholesterol in the diet as adults, and have little capacity to eliminate it when it is fed to them in large amounts. Other animals, such as the rat, the dog and man, have much greater ability to handle it and are relatively unaffected by dietary administration. Much has been made of the fact that when rabbits and chickens are fed diets of high cholesterol content they show a high concentration of cholesterol in the serum, sometimes more than ten times the normal value, and they develop atherosclerosis. But the amounts of dietary cholesterol used in these experiments are, in proportion to Calorie intake, fifteen or twenty times what is considered a high cholesterol diet for man. Rabbits and chickens do not normally meet cholesterol in their ordinary diets, and the metabolism of cholesterol in these animals is very different from that in man. In man, on the other hand, feeding very large amounts of cholesterol, 20 or more grammes per day, has only a trivial or no effect on the serum cholesterol concentration even over some months. Individuals vary greatly in their intake of cholesterol in their diets—from less than 100 milligrammes to one gramme per day. Results of examination of 35 middle-aged men who regularly consumed diets of very high cholesterol content were compared with those of 51 men who consumed diets of very low cholesterol content over a period of two years. There was no appreciable difference in the average serum concentration of cholesterol between the two groups. A number of other experiments giving similar results could be quoted. On the other hand, the total fat content of the diet has a pronounced effect on blood cholesterol levels. Feeding men on a diet of very low total fat content immediately initiates a decline in serum cholesterol levels, which in three weeks may have fallen to 70% of those associated with the ordinary free diet. Adding extra cholesterol to the diet makes no difference in the results. In order to determine the effects of normal dietary variations, Keys, with the assistance of collaborators in each country, examined the blood of groups of men in London, Naples and Madrid, and compared these with figures obtained in Minnesota in relation to differences in diets. Because of low consumption of eggs, meat and milk in Britain, the cholesterol content of the diet was much lower than in Minnesota, but the total fat intake was little different. The serum cholesterol figures were very similar. In Naples the average fat consumption was less than half that in Minnesota, and the serum cholesterol concentrations, instead of rising steadily after forty years of age, fell slightly and kept at the lower level with increasing age. In Madrid poor men with little fat in the diet and wealthy men with a high fat diet were studied; the poor men had low serum cholesterol levels at all ages, but those for the wealthy men were not very different from the Minnesota standards. Keys has also studied the mortality from degenerative heart diseases in various countries in relation to the average dietary intake. There is a notable correlation between the fat Calories, as a percentage of the total Calories, and the deaths per thousand from degenerative heart diseases (coronary disease, *angina pectoris*, infarction, chronic myocarditis and myocardial degeneration) in the age group fifty-five to fifty-nine years in the years 1948-1949. Thus Italy, with 20% of the Calories as fat, had fewer than two deaths per thousand; England and Wales, with about 30·3% of fat Calories, had about four deaths per thousand; Australia, with about 30·7% of fat Calories, had 5·6 deaths per thousand; the United States with 40% of fat Calories had seven deaths per thousand.

From this very thorough study several points become clear. None of the determinations of blood constituents is of any value in an individual case as an aid to diagnosis of atherosclerosis, although several of them are very useful in studying groups of people. The total cholesterol concentration of the serum of man is substantially independent of the dietary cholesterol intake over the whole range of possible human diets. The cholesterol content of the serum is very closely related to the total fat intake in the food, and there is no evidence of a difference between animal and vegetable fats in this regard. Comparison of men from populations always living on different amounts of total fat intake indicates that in youth and to the early thirties the serum cholesterol concentration is relatively independent of the diet if there is caloric sufficiency. But from the thirties through the fifties the serum concentration is increasingly dependent on the amount of total fat in the diet, and the same relations hold for the deaths of men from degenerative heart diseases. The relationship for women is much less clear. That there is serious disturbance of cholesterol metabolism in atherosclerosis seems to be clear, but how the fat intake affects this is not at all obvious. That there are other factors concerned is certain, for many people regularly consume a diet of high fat content and do not develop atherosclerosis. Still, the case against a high fat intake is so good that it seems to be wise to recommend that men over forty years of age should not increase their total fat intake as they commonly do, but should rather decrease it. Many men of middle age, and over, eat well over 100 grammes of fat per day. In such a diet two ounces of butter would provide about 46 grammes of fat, one egg 5·5 grammes, and one half-pint of milk 11 grammes; so for reduction we must look mainly to other sources of fat, particularly fat used in cooking. There seems to be no acceptable reason for the condemnation of eggs and dairy products.

Current Comment.

EFFECTS OF SMOKING ON NUTRITION.

ALTHOUGH an enormous number of papers have been written on the effects of tobacco smoking on bodily functions and the incidence of disease, very little experimental work has been done on the effect of habitual smoking on nutrition. J. M. Walker[1] sums up the available evidence in a paper given at a meeting of the Nutrition Society in Aberdeen in January, 1953. As he points out, it is a

[1] *Proc. Nutrition Soc.*, 1953, Volume XII, Number 2.

common observation that some middle-aged persons, who suddenly stop smoking, rapidly gain in weight. This could be due to decreased metabolic rate, a lower level of bodily activity, an increased absorption from the intestinal tract, an increased food intake or a combination of two or more of these factors. While the first three cannot be excluded, little can be said about them. Smoking does increase the basal metabolic rate in some persons, probably by liberating adrenaline, but the effect is slight and transient. There is no evidence that smoking increases bodily activity, or that cessation of smoking makes an individual more torpid. Smoking has no important effect on gastric secretion or emptying time, and enzyme activity is not reduced. We are left then with the suggestion that smoking reduces the appetite. The so-called hunger contractions of the stomach are reduced; but we know now that the stomach has very little to do with appetite and hunger, and it seems certain that hunger depends largely on blood sugar level. It has been claimed that smoking raises the blood sugar level, but this is back to normal within half an hour. The raising of the blood sugar level is probably due to adrenaline secretion, which is known to occur during smoking. Walker suggests that tobacco, or rather nicotine, acts directly or indirectly on the hypothalamus. Amphetamine is very effective in reducing appetite. This inhibits gastric contractions in large doses, but effective doses can be small enough to have no effect on the stomach. It does not cause a rise in blood sugar level. Recent work indicates that there is a centre in the hypothalamus which controls appetite and is affected normally by increase or fall in blood sugar level. It seems reasonable to suppose that this drug acts on the central nervous system and in particular on this part of the hypothalamus. A number of the other amine drugs also cause loss of appetite, and these are all drugs which inhibit the action of amine oxidase and thus protect adrenaline from destruction. Nicotine liberates adrenaline. Cigarette smoking inhibits water diuresis in man, and the injection of an appropriate amount of nicotine has the same effect; but what relation to nutrition this may have we do not know. The effect of smoking on body weight is of practical importance, but it is not clearly understood.

SUDDEN DEATH DURING INFANCY.

EVERY practising member of the medical profession must be able to produce from his recollection a number of disturbing cases of sudden death of young infants, in which the cause was obscure. One of the features of these distressing occurrences is the suspicion that some form of neglect may have been related to the child's death, whereas experience has shown that infective disease of the fulminating type known to occur in the young may be the cause. It will be remembered that K. M. Bowden pointed out in an article published in this journal that the alleged accidental suffocation which is not infrequently the subject of discussion in coronial inquiries warrants careful pathological investigation.[1] In this and in the second series published by Bowden and E. L. French the importance of acute pulmonary infections was made clear.[2] This same subject, which has produced a considerable literature, is one of those in which unbiased scientific inquiry is much in demand, for it is medico-legal in scope and should have all vestiges of popular misconception cleared away. Jacob Werne and Irene Garrow have recently published a series of 31 unselected cases of infant deaths occurring in New York City, and in a clear text and with excellent illustrations set forth the findings.[3]

Werne and Garrow state, as others have before, that the allegation of accidental mechanical suffocation by posture or bedclothes is unsupported by any real evidence. They have tabulated their cases and correlated the clinical history with the carefully performed pathological research which is necessary in such happenings. The autopsy findings are described for every system of the body, representative detailed studies are used to illustrate the outstanding lesions, and coloured and black and white plates give the microscopic proof for the diagnosis. In their summing up the authors point out that where a routine of full inquiry has been established the frequency of apparent suffocation has been greatly reduced. They quote Bowden and French's findings, which showed a high proportion of tracheo-bronchitis and acute bronchitis. In their conclusions they report the constant finding of inflammatory lesions in the upper and lower respiratory tract, where no other cause was found, together with vascular lesions in the respiratory tract and other organs. In the lymphatic tissues there were conspicuous changes, such as hyperæmia, hæmorrhages, thrombosis, destruction of lymphocytes and the presence of cellular debris in Hassall's corpuscles. Plasma cells were often seen, and the demarcation of the white from the red pulp of the spleen was partly lost. In a general discussion the authors point out that the general pattern of the pathological changes found in infants who had died suddenly was quite different from those observed after any form of violence which was believed to be the true cause of death. They like other writers, comment that smothering has been a kind of traditional cause assigned for death occurring suddenly in infants while apparently in good health. The obvious answer is that careful pathological examination has shown that these infants were not actually in good health, and the suddenness of illness and even death in babies, who are so susceptible to acute respiratory disease, must be taken into consideration. It is interesting to read that Brouardel, a French authority who wrote of sudden death 57 years ago, describes how young children may recover from a first attack of "congestion" or may succumb, especially if under six months of age. If such a child, he remarked, was found dead, particularly if the mother was an unmarried girl, suffocation would be suspected, whereas an autopsy would reveal the "capillary catarrh" of Laennec. The present authors propose to publish two more papers dealing with the same subject in different aspects; even more light will be welcomed, for there still seems to be a need. In country districts in particular a local practitioner has sometimes to decide questions involving not a little pathological judgement: sudden death in infants is one, but responsibility may be shared, and it is both simple and desirable to submit tissues for further examination.

[1] M. J. AUSTRALIA, January 21, 1950.
[2] M. J. AUSTRALIA, June 30, 1951.
[3] *Am. J. Path.*, July-August, 1953.

CHAIRS AND SITTING.

VARIOUS factors are taken into account in the design of chairs. Elegance and æsthetic qualities generally exert a good deal of influence, particularly in relation to more expensive chairs. Certain traditional ideas on what is likely to be comfortable help to determine the design of lounge chairs, theatre seats and the like. The straight-backed chair is still preferred by many people, either because, in its more elaborate form, it follows some classical design acceptable to its owners, or because it is thought to encourage a physiologically correct sitting position. The fact that seems to have received least attention is the accurate determination of what is physiologically sound. Valuable data on this subject have recently been presented by J. J. Keegan[1] in an analysis of the subject of seating in relation to back symptoms. This work is based on a clinical study of over 3000 persons with low back complaints, 1504 of whom have been operated upon for herniation of a lower lumbar intervertebral disk, as well as on a special study of the alteration of the lumbar curve of subjects in various sitting and standing positions. Keegan points out that one of the most common complaints in persons with low back pain is inability to sit in comfort, with difficulty in straightening the back on rising. This is particularly noticeable after sitting for a long time in a lounge chair, or an automobile or theatre seat, all of which are supposed to be comfortable. Keegan suggests that this common complaint must represent some funda-

[1] *J. Bone & Joint Surg.*, July, 1953.

mental defect in our conception of the correct sitting position and in the design of chairs and seats, for only young persons with elastic ligaments and no pain in the back can tolerate sitting for long in the type of seats commonly designed. Older people, who use chairs more often, do not have this elasticity and often sit in discomfort. Keegan refers to a recent monograph by Akerblom, in which the anatomy and physiology of the problem of sitting are presented, but goes on to comment that there is still a lack of understanding of the pathological causes of most postural low back pain related to seating. He considers that recently acquired knowledge of the pathology of lower lumbar intervertebral disk lesions should be applied to the seating problem, and that more correct fundamental rules should be presented for the design of seats for the many persons with low back complaints. These rules, it is interesting to note, are described as being equally applicable to normal persons. In a series of striking illustrations Keegan presents evidence from tracings of skiagrams from four normal young persons in order to show the normal physiological alteration of the lumbar part of the spine when the subject is in various positions. It is illuminating to see how various positions, particularly as they are determined by various types of seats, influence the structures of the lower lumbar spinal region, including the fourth and fifth lumbar intervertebral disks; it adds significance to Keegan's statement that the site of most back symptoms arising from postural factors is in this region, and particularly in the disks which commonly degenerate with age under normal weight-bearing, sitting and stooping strain. In middle-aged people, some loss of elasticity has occurred in intervertebral disks and ligaments, and obliteration of the lumbar curve tends to force the degenerated and somewhat separated central portion of the lower lumbar disks posteriorly by hydraulic pressure from anterior wedging. This occurs in variable degree during sitting, and causes painful stretching of the sensitive posterior longitudinal ligament of the disk, with pain in the midline and the lower part of the back. When the subject is in the stooping position, particularly during lifting, this hydraulic pressure is greatly increased; this may cause rupture of the disk with extrusion or herniation of the loose central fibrocartilage postero-laterally, and consequent nerve root pain due to pressure, and usually disappearance of the original back pain.

Many common designs of chairs and seats fail dismally to maintain the lumbar curve in a satisfactory state. Keegan's study indicates that the optimum or physiologically normal position of the adult spine is with the trunk-thigh angle and the knee angle at approximately 135°, and that in this position there is considerable curve in the lumbar part of the spine. This is the position of balanced muscle relaxation with the intervertebral disks stabilized in their normal adult shape. Alteration of this normal adult lumbar curve either forward during sitting and stooping, or backward during standing erect, places greater wedging strain on the lower lumbar disks than on those above, because of the tensing of the long thigh muscles of the opposite side and fixation or rotation of the pelvis. Sitting positions which tend to flatten or obliterate the lumbar curve by decreasing the angle between the thighs and the trunk soon become uncomfortable and cannot be tolerated for long by persons with degenerated intervertebral disks in the lower lumbar region.

With the basic knowledge revealed in this study, Keegan has prepared an outline to show the correct rules for the design of chairs which he considers applicable to all seats, regardless of their purpose, size or cost. He states that the most important requirement of a correct seat for protection of the vulnerable lower intervertebral disks is placement of the primary back support over the lower lumbar region. Free space or a sharply recessive curve is then needed below the lumbo-sacral junction to accommodate the posteriorly projecting sacrum and buttocks. With small straight chairs the back support should end at the thoraco-lumbar junction, below the lower angles of the scapulæ. Shoulder support is necessary only in chairs for semi-reclining and should always be made secondary to the primary support of the lower lumbar curve. The next important requirement for a correct seat is to allow a minimum trunk-thigh angle of 105° in high-backed chairs. The seat of the chair should be short, never over 16 inches in length, the length being reduced as the seat is made higher. The seat should not extend beyond the mid-thigh region beneath the tendons at the back of the knees. The front border of the seat should be rounded and narrow, and there should be an open space beneath, so that the feet may be placed beneath the seat for relaxation and as an aid during rising. The height of the seat from the floor may vary according to purpose, the seat necessarily becoming shorter as the distance from the floor is increased, so that the thighs can be directed more downward and the feet can reach the floor. Another important point is that it should be possible for position to be changed in the seat. A number of other factors desirable in seating are mentioned by Keegan, and we may look forward with interest to a promised subsequent article which is to deal with the subject in a more specific manner, including discussion of the faults of types of seats in common use and presentation of correct seat design. The possibility of educating chair manufacturers and, more important, their customers in sound seat and chair designs is another matter.

CHEMOTHERAPEUTIC MANAGEMENT OF INFANTILE DIARRHŒA AND VOMITING.

A CARDINAL principle in the management of infectious diseases with antibiotics and chemotherapeutic agents generally is the determination of the organism responsible and the selection of the therapeutic agent to correspond with that organism. However, the rigid application of this rule breaks down in the treatment of infantile diarrhœa, as only too often a causative organism cannot be isolated. This point has been emphasized by many different workers. For example, Rose Mushin[1] has stated that, with the exception of hospital outbreaks, the recognized pathogens such as Salmonella and Shigella are responsible for a relatively small percentage of cases of diarrhœa, especially in temperate climates. Other suspected agents range from viruses and bacteria to fungi and protozoa, but there is still a large anonymous group left for investigation. One should also consider the dietetic and psychosomatic factors which may be involved as predisposing conditions towards infectious diarrhœa, but the search for the elucidation of the unknown factors still continues. Sydney D. Rubbo[1] points out that a fuller understanding of the epidemiology of infectious diarrhœa is contingent on a better understanding of its ætiology. It is not improbable, he states, that our rigid views on what constitutes an enteric pathogen may require reorientation, and our casual interest in the lactose-fermenting coliform bacillus, because it is invariably found in the normal alimentary flora, may be a dangerous premise. Speaking in a symposium on the subject of diarrhœa at the Australasian Medical Congress (British Medical Association) in Melbourne last year, V. L. Collins[2] said that over the previous twelve months 350 patients with entero-colitis had been admitted to the Children's Hospital, Melbourne; 56 had a Shigella infection, 51 a Salmonella infection, and 243 non-specific infections.

It is generally agreed that, whatever associated factors may be concerned, the so-called non-specific infantile diarrhœa and vomiting are in many instances manifestations of some acute infection. The absence of information on the causative organism has not prevented the use of sulphonamides and antibiotics for therapy on an empirical basis, and assessment of their value on clinical grounds. At the same time, the prognosis associated with infantile diarrhœa and vomiting has greatly improved, a change contemporaneous with the adoption of more effective measures for the treatment of dehydration. Results attributed to particular drugs need therefore to be considered in relation to improvement in current methods of

[1] M. J. AUSTRALIA, March 29, 1952.
[2] M. J. AUSTRALIA, October 18, 1952, page 570.

routine treatment. Many of the published results are difficult to assess; so with this in mind therapeutic trials were carried out by a working party established by the Antibiotics Clinical Trials (Non-Tuberculous Conditions) Committee of the Medical Research Council of the Privy Council.[1] The trials were designed to investigate the clinical effects of the oral administration of aureomycin, chloramphenicol and sulphadiazine on infantile diarrhœa and vomiting, excluding Salmonella and Shigella infections. Following the admirable system adopted in many Medical Research Council investigations, the work was undertaken at a number of separate centres, ten in all, in various parts of Great Britain. It began in March, 1950, and continued for about two years. The investigation was controlled, although the method of control had to be modified considerably, with every justification, when ethical considerations forbade the withholding of the treatment under trial from children of the control group who were not responding to other methods of treatment. All drugs were administered by mouth, the dosage being as follows: aureomycin 75 milligrammes, chloramphenicol 75 milligrammes, sulphadiazine 125 milligrammes, in each case per pound of body weight per day for seven days. A total of 1168 cases (789 mild, 379 severe) was included in the analysis of the results. Of the 1168 patients, 352 were controls, 154 were treated with aureomycin, 415 with chloramphenicol and 247 with sulphadiazine. Clinical progress after the commencement of treatment was assessed on the following criteria: average duration of diarrhœa, average time before full clinical recovery took place, and proportion of mild cases which became severe. It was found that progress in the aureomycin-treated group was more favourable than in the control group as regards the proportion of mild cases which became severe; but it was not more favourable as regards the other two criteria. Patients in the chloramphenicol-treated and sulphadiazine-treated groups progressed more favourably than those in the control group as regards all three criteria. The progress of those in the sulphadiazine-treated group was significantly better than that of those in any other group.

The report points out that in the assessment of the results of the investigation as a whole it should be emphasized that all the trials were made against a background of highly effective supportive treatment. The fatality rate was 2·8% for the whole group of 1168 patients, and even in the control group it was not higher than 4%. The number of deaths in the control group may have been influenced by the use of chemotherapeutic or antibiotic agents which were given to improve their chances of recovery to some infants whose clinical condition was deteriorating. For this reason the fatality rate was considered an unreliable criterion for the comparison of trial and control groups. However, the differences between the fatality rates in the various treatment groups, though not large enough to be statistically significant, were consistent with the differences observed as regards the other criteria of clinical progress. More than half the controls made straightforward progress to recovery without chemotherapeutic or antibiotic treatment. Nevertheless, the proportion of mild cases becoming severe was significantly greater in the control group (14%) than in the aureomycin-treated (4%), chloramphenicol-treated (3%) or sulphadiazine-treated (4%) group. The working party considered that this finding suggested an important use for these drugs in the prevention of clinical deterioration in mild cases when the patient was not responding readily to routine methods of treatment. Moreover, the administration of sulphadiazine or of chloramphenicol, but not aureomycin, was shown to reduce the average duration of illness in both mild and severe cases.

These are broad findings from what can be described only as an investigation of empirical treatment; but, as the report points out, selection of the most appropriate drug for treatment in individual cases of infantile diarrhœa and vomiting may become possible when more is known of the ætiology of the disease. The results of the present investigation indicate that sulphadiazine and chloramphenicol are the drugs of choice, with the balance of evidence in favour of sulphadiazine, and their oral administration may be considered as a valuable addition to established methods of treatment. However, the report very rightly stresses the fact that adequate supportive treatment still remains of paramount importance. The administration of sulphadiazine or of chloramphenicol cannot be regarded as in any sense a substitute for this basic therapy, and the risk of toxic manifestations from the use of either of these drugs must be constantly borne in mind.

[1] *Lancet*, December 5, 1953.

LACTATION AND FLUID INTAKE.

MEDICINE has its quota of ideas that nobody ever thinks to question. Occasionally such an idea on close scrutiny is found to be a complete impostor and has to make a hurried retreat from the scene. The latest idea to be exposed and apparently disgraced is the custom of advising mothers to drink a lot of fluid to increase their milk supply. The exposure comes from R. S. Illingworth and B. Kilpatrick.[1] It followed the thought on the part of Illingworth that it was unlikely on physiological grounds that mothers would produce more milk if they took more fluid than they felt the desire to take. It seemed likely to him that thirst, often experienced by the mother when the baby begins to suck, was Nature's method of seeing that fluid needs were supplied in lactation, and that additional fluid was unnecessary. As a result a controlled investigation was carried out on the relationship of fluid intake to the amount of milk produced by 210 mothers in a maternity unit. Half the group of mothers (106 to be exact) were given no instruction about how much fluid they should take, being left to drink what they wanted; the average daily amount of fluid drunk by them was found to be 69·1 ounces. The other half, or 104 mothers, were instructed to take at least six pints of fluid a day; the average daily quantity taken by them was found to be 107·5 ounces. The gain in weight of the babies, the result of test feeds and the incidence of full breast feeding were observed, the babies being divided into three birth weight groups. No evidence was obtained that the forcing of fluids had any beneficial effect on lactation. On the contrary, there was evidence that more milk was produced by mothers who drank only enough to satisfy their needs. When the amount of fluid taken by all the mothers was correlated with the test feeds, there was a significant negative correlation coefficient in favour of letting the mothers satisfy their fluid needs without being asked to take more fluid.

After the investigation had been completed, a search was made for other work on the subject, but only a single investigation described in two papers could be found. The papers were both by A. Olsen, and appeared in Scandinavian medical journals. There it was pointed out that if it was true that forced fluids increased milk production, one might also expect a corresponding increase of other body secretions, such as tears and saliva, when additional fluids were taken; that was not known to happen. Olsen remarked that there was no evidence that cows produced more milk when given extra water to drink. In a careful study of thirteen nursing mothers he had been unable to find any correlation between the amount of fluid taken by the mothers and the amount of milk produced, and he reached the following conclusion: "Forced excessive drinking is therefore neither necessary nor, as a rule, beneficial as far as the nursing is concerned, and may even be harmful." This is in accordance with the more extensive and better controlled investigation by Illingworth and Kilpatrick, who would seem quite justified in their recommendation that the widespread custom of trying to persuade mothers to drink large quantities of fluid during lactation should be abandoned. They should merely take enough to satisfy their own desires.

[1] *Lancet*, December 5, 1953.

Abstracts from Medical Literature.

THERAPEUTICS.

The Emergency Treatment of Cardiac Arrest.

ROBERT M. HOSLER (*J. Internat. Coll. Surg.*, March, 1953) states that sudden heart stoppage can happen during a so-called simple operation as well as during a complicated one. It can take place with the patient under local or regional anæsthesia as well as under general anæsthesia. It would seem to occur in patients without known heart disease more frequently than in those whose hearts are known to be affected. When it occurs in a person with a good heart and a good pair of lungs, the author likens it to the turning off of the ignition key in an expensive automobile. Theoretically, the car is as good as ever, but unless its motor can function, it is as worthless as a pile of iron. In a case of cardiac arrest, to carry the analogy further, the surgeon turns on the ignition by supplying oxygen and cranks up the motor by manual massage of the heart. Reliable figures on the incidence of this operating room catastrophe are not available. An estimate now commonly cited is that sudden cardiac standstill will occur, on an average, five times a year in a 700-bed active hospital. Fundamentally, the ætiology is a lack of oxygen in the vital tissues or inadequate elimination of carbon dioxide. This statement is not very specific, but in some instances the specific factor can be pinpointed. However, one or more of the following predisposing factors may be responsible: decreased vital capacity, tracheal obstruction, poor oxygenation, anæmia, anxiety, insufficient atropine, shock, poor cardiac filling, torsion of the heart, excessive accumulation of carbon dioxide in spite of good oxygenation, excessive number and quantity of anæsthetic drugs and apathy. The author discusses the use of a defibrillating machine, and advises the need for constant alertness and adequate equipment. Frequent rehearsals or "fire drills" are suggested. The surgeon may be confronted with either of two conditions of cardiac arrest in the operating room, namely, cardiac standstill and ventricular fibrillation. The first action is the placing of a tube in the trachea and delivery of oxygen to the lungs under positive pressure. Then the surgeon boldly and quickly opens the left side of the chest, places his hand on the heart and pericardium and institutes cardiac massage. If the heart is not beating rhythmically, the next step is to open the pericardium widely and immediately resume massage. The author gives detail of this procedure and discusses the use and abuse of drugs.

Drug Idiosyncrasy and Sensitization.

G. DISCOMBE (*Practitioner*, April, 1953) discusses drug sensitization and idiosyncrasy. He states that sensitization to drugs taken by mouth or injected is less common than sensitization to drugs applied to skin surfaces. Drug sensitization affects the skin and the blood-forming organs most often; rarely asthma or hay fever appears. Most drugs may cause dermatitis, but so may synthetic detergents, white flour and *Primula obconica*. The antibiotics, sulphonamides and anæsthetic esters derived from para-aminobenzoic acid are the most important skin sensitizers. Streptomycin and dihydrostreptomycin may cause urticaria, erythema or exfoliative dermatitis. Penicillin may cause eczema when applied locally or injected. Benzocaine surface analgesics, used for *pruritus ani* or other conditions, may cause severe dermatitis. Blood dyscrasias may be due to drugs, which the author lists. He states that in the prescribing of any of the drugs great restraint should be used; the patient should be told to report any symptoms such as headache, sore throat or fever, and should then have a leucocyte count performed. Even a daily leucocyte count cannot exclude the chance of agranulocytosis with a considerable number of well-known drugs.

Dimercaprol (BAL) Ointment and Chronic Chrome Dermatitis.

H. N. COLE (*Arch. Dermat. & Syph.*, January, 1953) states that chromium compounds affect the tissue proteins. In strong enough concentration they corrode the skin and mucous membranes. Strong solutions not concentrated enough to corrode the skin may produce toxic dermatitis. In still weaker concentration, the chromates may sensitize the skin and cause allergic dermatitis. Chrome dermatitis is common. There is no specific treatment. The problem is best approached by methods of prophylaxis. These include the wearing of long rubber gloves and the use of protective ointments. The author decided to use dimercaprol in a concentration of 3% in zinc oxide paste on account of Salzberger's experience in demonstrating the sensitizing potentialities of a 5% concentration. The results of a small-scale animal study are presented, which shows 25% to 50% improvement within one week in animals with experimentally produced chrome dermatitis, treated with 3% dimercaprol ointment. Seven cases are presented in which patients suffering from chronic chrome dermatitis were treated with 3% dimercaprol ointment. Intolerance to dimercaprol was noted in one case. Two patients in hospital with severe dermatitis showed complete clinical remission within a period of two weeks with the daily application of 3% dimercaprol ointment to the affected areas. The four remaining patients still exposed to chromates have shown up to 75% improvement with continued daily use of the ointment for periods ranging from two to five months. The author suggests the use of 3% dimercaprol ointment in cases of chronic heavy metal dermatitis, particularly chrome dermatitis. The patients should be in hospital to get the best results.

Sandpaper Abrasion Treatment of Tattoos.

E. A. STRAKOSCH (*Arch. Dermat. & Syph.*, January, 1953) states that heretofore the removal of tattoos has been carried out by the plastic surgeon; various chemical and mechanical means have also been used, with resultant scar formation, but none of them have been satisfactory. The author reports three cases in which treatment was by abrasion with sandpaper. The sandpaper was rolled around gauze rolls about two inches in diameter and five inches long, kept in place by rubber bands, and autoclaved. The area was abraded, with circular motion and considerable pressure; the abrasion started from the centre and moved towards the periphery, until bleeding was profuse. The blood was immediately and thoroughly washed off with a gauze sponge soaked in warm saline solution containing adrenaline. The author states that this is important in order to keep the operating field clear and remove all grit particles, which would later lead to foreign body reactions. After removal of the tattoo marks, as verified by a magnifying glass, an aureomycin gauze pressure bandage was applied and kept in place for ten days.

Oral Penicillin Therapy for Pyogenic Skin Infections.

J. M. FLOOD (*Arch. Dermat. & Syph.*, January, 1953) sets out to show the effectiveness of orally administered penicillin in the treatment of pyogenic dermatoses. The patients selected for treatment had clinically demonstrated pyogenic infection, and bacterial cultures were obtained in all but one of the 16 cases observed. Penicillin was given in varied dosage. The author considers that the average adult dose would be 400,000 units three times a day for three days and then 200,000 units (one tablet) three times a day for five to seven days. Fifty tablets would probably control the majority of such infections in adults. For children, a soluble tablet containing 100,000 units of penicillin G was used; the total dose given was about half that for adults. In all cases except one the response was immediate, and pyogenic infection was well under control in three or four days. Therapy was continued in reduced dosage beyond this time in order to prevent a relapse. The author states that since penicillin for oral use costs approximately one-half as much as other oral antibiotics, its use is of advantage to the patient.

Intravenous Use of ACTH.

ERIC L. SMITH (*Arch. Ophth.*, March, 1953) reviews the literature on the intravenous use of ACTH and in particular its use in ophthalmology. Analysis of the literature reveals that the hormone can be best administered in 500 to 2000 mils of either 5% glucose solution or isotonic saline, which is given over a period of eight to twenty-four hours. Salt restriction and the addition of 0·2% potassium chloride solution are recommended. Continuous intravenous administration of ACTH results in an enormous increase in secretory activity of the adrenal cortex, as compared with the intermittent stimulation which results from the usual method of intramuscular injection. When 20 milligrammes of ACTH are given intravenously each day to a normal patient, a maximum response is attained in from three to eight days. Common complaints during the first few hours of infusion are flushing of the face, a feeling of tightness across the chest and a sense of fullness in the abdomen. The slower the rate of infusion, the less frequent these complaints become. Intravenous administration of ACTH is cheaper than the intramuscular method. The author reports on the use of intravenous ACTH therapy in a case of optic neuritis in

which the outcome was favourable; there had been no improvement with oral cortisone therapy. He concludes that intravenous administration of ACTH is undoubtedly a most valuable addition to many other forms of therapy of optic neuritis.

NEUROLOGY AND PSYCHIATRY.

Sleep Paralysis Treated with Insulin Hypoglycæmia.

H. A. WEITZNER (*Arch. Neurol. & Psychiat.*, December, 1952) describes a case of "sleep paralysis" successfully treated with modified insulin. He gives details of the technique of treatment and a very full clinical account of his patient. He comments that although the pathology of sleep paralysis is obscure, it is probable that the disturbance is a reversible physiological one. He suspects that the hypothalamus is the seat of the disorder and compares sleep paralysis with narcolepsy (which has likewise been treated with insulin hypoglycæmia). He discusses the role of the hypothalamus and sleep, and various possible ways in which the hypoglycæmia may influence the hypothalamic centres. His patient had a complete remission, lasting at least nine months and commencing promptly after the first treatment.

Peripheral Nerve Lesions in Hæmorrhage Diseases.

N. H. BIGELOW AND R. W. GRAVES (*Arch. Neurol. & Psychiat.*, December, 1952) report two cases in which hæmorrhage disease was accompanied by peripheral nerve lesions. In one case secondary thrombocytopenic purpura was produced by administration of penicillin; in the second case the rare primary hæmorrhagic thrombocythæmia occurred. In the former case there was impaired femoral nerve function with spontaneous hæmorrhage within the nerve. In the latter case the nerve injury apparently resulted from the pressure effects of a hæmatoma impinging upon the nerve.

Gerstmann Symptoms following Electroconvulsive Therapy.

A. L. BENTON AND L. S. ABRAMSON (*Arch. Neurol. & Psychiat.*, August, 1952) report five cases of psychosis treated with electroconvulsive therapy in which were observed Gerstmann symptoms (finger agnosia, right-left disorientation, agraphia and acalculia). The symptoms appeared after the cessation of convulsions when the patient was normally personally orientated. The authors state that the current concept of the ætiology of the Gerstmann syndrome is one of parieto-occipital dysfunction, being a single basic impairment of *Gestalt* formation. The authors attempted to verify this concept and examine the theoretical principles proposed as explanations for the association of these rather diverse behavioural deficits. A full description of the examination in their five cases follows, and they are able to state that their findings fully confirm the contention that Gerstmann symptoms follow electroconvulsive therapy, but that evidence for a general or *Gestalt* factor is doubtful. They are of opinion that the four disturbances of behaviour are related topographically and that their occurrence as a "syndrome" is explained in terms of proximity of cortical "centres" rather than in terms of basic psychological relationships.

Brain Lesions Associated with Prolonged Unconsciousness.

J. D. FRENCH (*Arch. Neurol. & Psychiat.*, December, 1952) describes five cases of disturbed consciousness due to various causes including cerebral thrombosis, head injury, cerebral anoxia and meningo-encephalitis. He analyses and compares these cases and gives a full clinical description and an account of autopsy findings. In three of the cases the major pathological lesion destroyed the main portion of the reticular activating system (facilitatory reticulum) in the cephalic portion of the brain stem. In the other two cases the lesions were more cephalad, in the basal ganglia and the entire cortical mantle. The author discusses his observations in the light of recent work on the importance of the brain-stem reticular formation in maintaining awareness and the conscious state. He concludes that the brain-stem activating reticulum is capable of exciting subcortical structures such as the thalamus and hypothalamus to activity, this permitting waking and sleeping in a crude sense. It also influences the cortex and is responsible for diurnal "wakefulness" and behaviour characteristic of the "conscious" adult. Injury to the reticular system renders the cortex impotent and produces stupor or parasomnia, without actual disturbance of crude sleep rhythms; total destruction of the system is incompatible with survival and results in disappearance of the crude waking response and true coma. The author presents his five cases as illustrations of this theory.

Familial Spastic Paraplegia.

G. A. SCHWARZ (*Arch. Neurol. & Psychiat.*, November, 1952) gives a detailed account of his researches into the pedigrees of four families whose members have spastic paraparesis or paraplegia. His study included the histories, neurological examinations and laboratory studies of 12 diseased members of the 32 persons said to have been afflicted in these families. The author describes also the pathological findings in the spinal cord in a case previously reported clinically by Bayley and Spiller. He states that he observed a demyelination of the cortico-spinal tracts and certain of the extrapyramidal spinal pathways. In conclusion the author attempts to suggest the direction of future pathological studies of similar cases, based on newer anatomical and physiological knowledge and more precise pathological techniques.

Intensive Treatment of Backward Patients.

E. F. GALIONI AND F. H. ADAMS (*Am. J. Psychiat.*, February, 1953) discuss two questions: (i) Will more adequate staffing with intensified use of common present-day treatment procedures permit some chronic mental hospital patients to return to the community? (ii) What is adequate staffing? An investigation on these lines was carried out. Four hundred chronic male patients were selected. Various group activities were carried out by the nurses, with the object of conveying to the patient that the staff was interested in the patients' general welfare and was prepared to give supportive help for the individual patient's needs. Such activities were reading, retelling of stories, outdoor exercises and games. In a control group of 200 patients, the staff was chiefly engaged upon administrative duties. The result showed that two and a half times as many people left hospital from the group having intensive help as from the control group, and there was a greater movement to return to the premorbid personality among members of the treated group. The cost of the intensive treatment was more than double. The authors suggest that interest in the patients' welfare should be emphasized and that the patients' families must be considered an essential part of the therapeutic programme. Effective community placement measures become essential to keep the improved patient out of hospital.

Mid-Brain Tumours.

M. G. NETSKY AND R. R. J. STOBAS (*Arch. Neurol. & Psych.*, July, 1953) report four cases of neoplasm in the mid-brain and review the literature, in which they have found reports of 19 cases. They state that the neurological signs most often encountered are paralysis of the conjugate vision, papilœdema, ataxia and pyramidal tract signs. Early and pronounced mental changes including a wide range of disturbances are common, and constitute a diagnostic trap. The triad of mental change, conjugate paralysis and signs referable to the long tracts is suggested as a diagnostic aid. The commonest tumours are astrocytoma and *spongioblastoma polare*. Air ventriculography is revealing but a risky procedure.

Military Performance of Marginal Neuropsychiatric Subjects.

WILLIAM A. HUNT, CECIL L. WITTSON AND EDNA B. HUNT (*Am. J. Psychiat.*, September, 1952) studied the medical and service records of a group of 537 marginal psychiatric subjects, all of whom had completed three years of military service. For the purpose of this study the division was made into the common categories of neurotic, schizoid personality, alcoholic, psychopath and low intellect type. The authors state that the neurotic group constituted a hospital but not a disciplinary problem. When, however, the neurotic was guilty of some offence, there was a disproportionate amount of alcoholism. The schizoid personality group provided both a hospital and a disciplinary problem, but the disciplinary problem was not alcoholic. The alcoholic group provided a disciplinary and hospital problem with leave infractions and insubordination prominent amongst the offences. The psychopath group followed the alcoholic closely, but emphasis was on leave infractions and particularly insubordination. The person of low intelligence tended to provide a hospital and disciplinary problem with leave infractions but not alcoholism. The study emphasizes the fact that a lower quality of military service must be expected from these marginal subjects, and that an added strain is imposed upon the medical and disciplinary facilities.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XCII.

TONSILLECTOMY.

TONSILLECTOMY is the most frequently performed of all surgical operations. It is often performed unsatisfactorily. While few authorities agree on the indications for tonsillectomy, all are nowadays agreed that if the operation is necessary, "the tonsil, the whole tonsil and nothing but the tonsil", to use the words of T. B. Layton, must be removed.

Indications for the Operation.

The indications for operation cannot be expressed more succinctly than has been done by Samuel J. Crowe: "The tonsils should be removed when they are the seat of chronic infection, which produces or threatens to produce bodily injury greater than the disability, discomfort and inconvenience of removal." It is not always the place of the laryngologist to decide whether tonsillectomy is or is not necessary. The patient's own physician may be, and generally is, in a better position to decide the question. A letter from the patient's own doctor advising that tonsillectomy is in his opinion indicated is of much greater value than examination of the pharynx on one occasion only.

Age Limits.

There are no definite age limits for tonsillectomy, but the operation is rarely indicated in people aged under three years or over sixty years. When the operation is necessary in the latter case, it is usually because of mechanical obstruction to deglutition from gross tonsillar hypertrophy.

Contraindications.

The operation is contraindicated in the presence of an acute upper respiratory tract infection. Hæmophilia, purpura, leuchæmia and pregnancy are absolute contraindications to tonsillectomy. In cases in which questioning reveals a past or family history of undue bleeding, the operation is indicated only when tests of the bleeding and clotting time of the blood are found to give normal results. The operation on persons suffering from neuritis, diabetes, severe anæmia or cardiac disease should be performed only on the recommendation of, and with the collaboration of, a physician.

Poliomyelitis and Tonsillectomy.

The relationship of tonsillectomy with poliomyelitis is a matter of concern, and it is difficult to correlate the statistical findings of various authorities.

The belief has been generally held that if a patient contracts poliomyelitis within three or four weeks of tonsillectomy, he is likely to contract the bulbar form. It would appear from one series of investigations that any relationship between tonsillectomy and the bulbar form of the disease is apparently coincidental, and it has been advised that there is no need to defer the operation during an epidemic of poliomyelitis. Moreover, the conclusion to be drawn from another series of investigations is that the bulbar form of poliomyelitis occurring after tonsillectomy is more frequent in non-epidemic than in epidemic years.

More recently it has been suggested that tonsillectomized subjects are more likely to contract the bulbar form of the disease, even as long as ten years after the operation, than is the untonsillectomized subject. This would indicate that the operation should be completely abandoned till it is found possible to control poliomyelitis.

The writer's opinion is that in the present light of medical knowledge of the subject, each case in which tonsillectomy is required must be judged solely on his own merits.

Pre-Operative Measures.

All patients, whether children or adults, must be admitted to hospital the night before the operation, so that they may have a full night's sleep. With children most of the unpleasant features associated with an anæsthetic—terror during induction and fear persisting afterwards—can be avoided by suitable premedication. For this purpose pentobarbital is the most satisfactory drug. It is uniform in its action, and idiosyncrasies are not common. The administration of an anæsthetic to a struggling, terrified child should be a thing of the past. Children between the ages of three and eight years, of average weight, should be given one and a half grains of pentobarbital the night before the operation and again one hour before the operation. For children aged eight years and upwards the pre-operative dosage is two grains. Adults are adequately sedated by three grains of pentobarbital both on the night of the operation and one hour before operation. A hypodermic injection of atropine, 1/150 grain for children and 1/100 grain to 1/150 grain for adults (depending on the age and physique of the patient), is given half an hour before the anæsthetic is administered. The administration of morphine as pre-operative medication for tonsillectomy under general anæsthesia is contraindicated.

Premedication for Tonsillectomy Under Local Anæsthesia.

If the patient or the surgeon desires the operation performed under local anæsthesia, adequate premedication is a decisive factor in controlling fear and nervousness. Local anæsthesia is reserved for adult patients only. Pentobarbital is given by mouth in the doses mentioned above for adults, both on the preceding night and one and a half hours before the operation. One hour before operation grain 1/6 to 1/4 of morphine with grain 1/150 to 1/100 of hyoscine, depending on the age and physique of the patient, is given hypodermically. Young and elderly subjects should be given the smaller dose of the two last-mentioned drugs. With pentobarbital, morphine and hyoscine premedication, the patient is euphoric but cooperative enough, and after the insertion of the Boyle-Davis gag and with the operating theatre darkened, many patients doze throughout the operation and the majority have a retrograde amnesia about the whole procedure.

Long-Acting Local Anæsthesia.

Until recently the only long-acting anæsthetic agent available was eucupine-procaine solution. This is put up ready in serum bottles of 30 millilitres' capacity. It gives post-operative anæsthesia lasting from four to six days. At the end of that time a stronger solution (1%) can be sprayed onto the fossæ and a further four or more days' anæsthetic effect obtained. More recently "Andolar" and "Efocaine" have been available. To be effective for tonsillectomy, larger amounts of these have to be injected than is recommended by the manufacturer, and the risk that the cough reflex may be abolished is ever present. As the anæsthesia can last up to fourteen days, this risk is not one to be regarded lightly. If pain occurs six days after the operation, the patient complains more bitterly than when pain has been present from the day of operation. However, when the anæsthetic effects last for ten days, the results are gratifying, since, as mastication is possible from the day of operation, healing of the fossæ is very rapid. The aim should be to give enough of the solution to produce long anæsthesia, but not enough to risk abolishing the cough reflex. However, judgement of this dosage is difficult, and indeed the amount necessary seems to vary with different subjects.

As much of the pain after tonsillectomy is connected with swallowing and tongue movements, most of the solution should be reserved for the lingual part of the fossæ. The solution is injected immediately after each tonsil has been removed and before hæmostasis is secured. If the injection is placed too superficially, painful sloughing occurs. Sometimes swelling occurs immediately after the operation in the submandibular area and lasts for about four days. The cause of this is not obvious, unless it is the result of placing the injection too deeply into the tonsil fossæ. Fortunately this is not a common complication. It is, of course, important to guard against injecting the solution into a vein.

Choice of Anæsthesia.

For children ethyl chloride and ether given on an open mask followed by ether given through a Boyle-Davis gag is a very satisfactory anæsthetic in competent hands. For adults, if the patient has responded well to the barbiturates, induction with ethyl chloride and ether is satisfactory. For nervous subjects induction by the intravenous injection of "Pentothal" is a most pleasant way of beginning the anæsthetic. However, intravenous induction should be used only by the experienced anæsthetist. Intubation of the trachea for maintenance of anæsthesia should be left to the discretion of the anæsthetist, but experience suggests that nitrous oxide and oxygen given through the intratracheal tube cause more bleeding during the operation than do air and ether.

Position of the Patient during Tonsillectomy.

Whether the operation is performed under local or general anæsthesia, the hyperextended position of the head is preferable. This is obtained by placing a small sandbag or firm pillow under the shoulders of the patient. This should not extend under the patient's neck, and should not be placed too far under the shoulders or over-extension of the head will result. Under local anæsthesia, the cough reflex being retained, extension of the head need not be so complete as it is with general anæsthesia. The position should be such that the larynx is on a higher level than the fauces, so that blood tends to flow into the mouth and not towards the larynx. However, if the head is over-extended the result will be that the epiglottis comes into contact with the posterior wall of the pharynx and obstructs respiration. This is of no consequence if the trachea has been intubated, but otherwise the surgeon should make certain that half an inch or more separates the epiglottis from the posterior pharyngeal wall. As the anæsthetist has unavoidably to hand over the custody of the upper air passages to the surgeon, the latter should be certain that an adequate airway is present after the Boyle-Davis gag has been inserted.

The Boyle-Davis gag may be used when the operation is performed under either local anæsthesia or general anæsthesia. When the former is used, the lips, palate and tongue should be sprayed with "Pantocaine" solution (2%), as well as the pharynx before the local anæsthetic agent is injected.

The points for injection are each pole of the tonsil and each pillar. After the tonsil has been lifted forward with the tonsil-holding forceps, a further injection is made into the fossa beneath the tonsil before the operation is commenced. The amount of solution injected should be generous, to display the tissue planes as fully as possible so that dissection is easy and rapid. Large amounts of weak anæsthetic solutions are preferable to small amounts of strong solutions. Half an ounce of 1% procaine solution which contains seven minims of one in 1000 adrenaline solution to each fluid ounce should suffice for both tonsils. A drier surgical field is obtained if after the injection the gag is removed and five or more minutes are allowed to elapse before the operation is commenced.

Technique.

The actual technique by which the tonsil is dissected is not of importance. What is most important is that each fossa should be completely cleared of tonsillar tissue, or recurrence of lymphoid tissue in the fauces is inevitable. The completely removed tonsil never returns.

The operation may be divided into four stages.

Exposure.

The Boyle-Davis gag is inserted in the mid-line and opened widely enough to give adequate exposure of each tonsil. The tonsil is grasped with the tonsil-holding forceps and moved towards the uvula to display the *plica triangularis* and the fold of the anterior pillar.

The Incision.

A first snip with straight tonsil scissors is made through the plica just behind the fold of the anterior pillar (Figure I). Still with the straight scissors, the incision is continued down to the tongue and up to the upper pole of the tonsil (Figure II). The grip of the tonsil-holding forceps is now altered to include the edge of this incision. The closed points of the scissors are inserted about one-eighth of an inch and then opened to reveal the grey, smooth capsule of the tonsil.

Dissection.

The lower blade of the tonsil-holding forceps is now inserted into the gap made by the opening of the points of the scissors and the full thickness of the tonsil is taken in the grasp of the forceps. A few swift strokes with the slightly opened scissors or a sharp tonsil dissector on the subcapsular connective tissue, which is tensed by the traction of holding forceps, will free the tonsil completely. If hard, fibrous strands of adhesions or well-developed tonsillar fibres of the tonsillo-pharyngeus muscle resist sharp dissection, these may be easily snipped by the scissor points.

The dissection is carried right up to the tongue, and the lingual pole is stripped to the mucosa, especially at the base of each pillar. In some cases it will be necessary to dissect lymphoid tissue past the tongue down towards the vallecula. The posterior pillar should be conserved in its entirety and left intact till the tonsil has been completely dissected down to the tongue. After this has been done the posterior pillar is incised along its whole length as close as possible to the tonsil, curved tonsil scissors being used. Leaving the posterior pillar till the end of the manœuvre not only preserves it completely, but allows it to deflect blood onto the hard palate, where it can be removed by suction and kept away from the epipharynx and pharynx.

The tonsil, now adherent only by the mucous membrane to the tongue, is taken into the loop of the tonsil snare wire, the beaks of the holding forceps being passed through the loop to hold the tonsil; the snare is passed down as closely to the tongue as possible and closed to remove the tonsil. The area at the base of each pillar is then examined. If necessary the tongue-piece of the gag is moved to the opposite side to facilitate this. If there is any lymphoid tissue present in these areas, the snare is reapplied or the fragment is grasped by the holding forceps and evulsed with a short, sharp jerk.

FIGURE I.

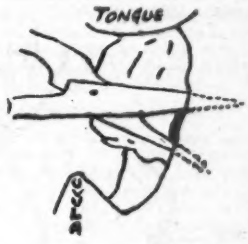


FIGURE II.

Hæmostasis.

The posterior pillar of the fauces is now drawn towards the mid-line by the holding forceps to facilitate the introduction of a tampon by a pair of hæmostats and then released. If a long-acting anæsthetic is to be used, it is injected at this stage before the tampon is introduced. The opposite tonsil is now dealt with in the same manner, after which attention is again directed to the tonsil first operated on.

The tampon is removed, the posterior pillar is retracted by the holding forceps and any bleeding points are secured and ligated. If no bleeding point is present, the "paratonsillar" vein should be ligated. If this is done, the chance of reactionary and secondary hæmorrhage is greatly reduced.

Ligating a bleeding point may present some difficulty after tonsillectomy. The use of the Coakley knot (Figure III) makes it possible with practice to ligate a point anywhere in the tonsillar fossæ. The knot is tied first and then applied with two hæmostats.

FIGURE III.

FIGURE IV.

The open tips of one curved hæmostat being used as retractors of the anterior pillar, the bleeding point is picked up by the other hæmostat and extended (Figure IV). At the same time the retracting hæmostat is reversed, and the tips, pointing to the mid-line, are slipped round the bleeding point extended by the other hæmostat (Figure V), which is removed. This leaves the bleeding point secured by a hæmostat with the point raised away from the tonsil bed to facilitate placing of the ligature. The knot is then placed over the hæmostat, the non-running part of the knot being held in the points of another hæmostat in the right hand. The free running end of the ligature is held

in the left hand (Figures VI and VII). The ligature is tightened as the bleeding point is released. Number three silk thread is a very suitable ligature for this type of knot, but must be quite dry from the autoclave or it will not run. A second knot can be tied over the running knot if desired, but it is quite unnecessary.

All bleeding points having been secured, the sucker is used to clear any blood that may be present in the epipharynx, on the hard palate or in the nostrils. The gag is removed and the patient is returned to bed, care being taken to maintain the airways if the patient is unconscious.

Post-Operative Care.

The patient, if he has had a general anæsthetic, is placed in bed on one side with the arm of that side drawn backwards. The opposite knee and thigh are flexed to support the trunk. The head is extended on the neck and the airway maintained. No pillow is used till the patient is conscious; but if the patient is an adult a pillow or sandbag, placed in front of the chest, helps to maintain the correct position.

FIGURE V.

FIGURE VI.

FIGURE VII.

When the patient becomes conscious, morphine may be given by hypodermic injection, the dose depending on the age and physique of the patient. After tonsillectomy under local anæsthesia, the patient is returned to bed with the head and shoulders raised.

Mastication as soon as possible after the operation should be encouraged. Patients who have a low threshold of pain can often be persuaded to chew by the prescription of one of the local anæsthetic lozenges now available to be sucked about a quarter of an hour before the meal. Similarly children have aspirin and honey. After the first day or so after the operation pain is lessened, especially in children, by giving chewing-gum a quarter of an hour before every meal. Some of the chewing-gum is inevitably swallowed, but seems to cause no ill effects. Food as solid as possible should be given; but if mastication is not attempted, semi-solid foods are given rather than liquids. Junket, jelly, blancmange and soup solidified by the use of gelatine and also ice-cream are generally easily taken, but occasionally one finds patients in whom ice-cream produces severe pain which may be referred to the ears.

Post-Operative Complications.

Reactionary Hæmorrhage.

Reactionary hæmorrhage is due to a bleeding point having been missed at the time of operation and not ligated, perhaps because the open Boyle-Davis gag had tightened the tonsil fossæ and temporarily controlled the hæmorrhage. When the gag is removed and the tissues are relaxed, the bleeding commences. This form of bleeding may be prevented by clearing all blood away after the operation is finished and then temporarily relaxing the gag. If any more blood collects in the fossæ, it means that there are more bleeding points to be attended to. If the bleeding is at all profuse after the patient returns to bed, the patient should be taken back to the operating theatre. If it is decided to wait, the pulse rate should be recorded every fifteen minutes; and if a rise is noted, return to the operating theatre should not be delayed. If a blood transfusion becomes necessary after a reactionary hæmorrhage, it means that the post-operative supervision of the patient has been inadequate. With a cooperative patient, oozing of blood can often be controlled by the removal of any blood clot and the introduction of a soft tampon moistened with adrenaline solution (one in 1000) with a tie attached.

Secondary Hæmorrhage.

Secondary hæmorrhage occurs between the sixth and tenth days and can in most cases be controlled by putting the patient to bed with the head and shoulders well propped up and giving ice to suck. If this fails, a hypodermic injection of morphine, the dose to depend on the age of the patient, should be given and the patient kept propped up in bed. It is rarely necessary even to remove blood clot from the fossa. Such patients do better when sent back to hospital, where anxious relations cannot disturb their rest after the morphine has been administered. Cultures prepared from the slough on the tonsil bed when there has been a secondary hæmorrhage generally yield profuse growths of hæmolytic streptococci. It is a good practice to give a large injection of procaine penicillin with or after the administration of the morphine.

Œdema of the Uvula.

Œdema of the uvula is occasionally seen after tonsillectomy, and on examination the uvula looks not unlike a peeled grape. Its occurrence is more common after the use of a long-acting local anæsthetic, and with the use of these it is wise to make a vertical incision in the lower part of the uvula to permit drainage. When the œdema has occurred the uvula is touched with a crystal or two of cocaine adhering to a wool-tipped applicator moistened with one in 1000 adrenaline solution. A dental or laryngeal mirror covered with gauze is placed behind the uvula, which is incised vertically. The œdema usually subsides within a matter of hours.

Partial Paresis of the Soft Palate.

Partial paresis of the soft palate sometimes gives rise to alarm in children, especially in those who cannot be persuaded to chew during the first four or five days after the operation. This is a rare post-operative complication and causes a "nasal" voice. It can persist for as long as twenty-one days. No treatment is necessary.

Parageusia.

Abnormality of the sensation of taste is a phenomenon seen rarely after tonsillectomy. It is more common in female patients and in patients who have undergone a previous tonsil operation. A "coppery", "metallic" or "sweet" taste along the back and side of the tongue is complained of, and is evidently due to the interference with fibres of the glossopharyngeal nerve, which supplies the mucosa of the tonsil. The sensation usually disappears in three or four weeks, but can persist for as long as six months. No treatment is required beyond reassuring the patient that the rather unpleasant condition will disappear.

Post-Anæsthetic Pulmonary Atelectasis.

Post-anæsthetic pulmonary atelectasis may occur as after any anæsthetic, and is treated by bronchoscopic aspiration.

Lung Abscesses.

The passage of blood or tonsillar debris into the trachea is said to be the sole cause of post-tonsillectomy lung abscesses. However, bronchoscopic examinations of patients who have undergone tonsillectomy half an hour previously have shown that blood is generally present in each bronchus. It is the opinion of Chevalier Jackson that the post-tonsillectomy lung abscess is not a result of blood entering a bronchus, but that the abscess results from a septic embolus carried by the blood vessels, causing a septic infarct.

The symptoms of a lung abscess are sudden in onset and may occur within three days after the operation and as long after as six weeks. When symptoms of fever, cough, pain in the chest or the expectoration of foul sputum occur after tonsillectomy, a radiographic examination of the chest and a bronchoscopic examination should be made immediately. Treatment by repeated bronchoscopic aspiration is effective in the great majority of cases.

HERBERT EARNSHAW,
Brisbane.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 18, 1953, at the Royal Alexandra Hospital for Children, Sydney. The meeting took the form of a series of clinical demonstrations by the members of the honorary medical staff of the hospital. Part of this report appeared in the issues of December 12, 1953, and January 2, 1954.

Hydatid Cyst in the Right Parieto-Occipital Region.

DR. T. Y. NELSON showed a girl, aged four and a half years, who had been admitted to the hospital on February 11, 1953, because of the following symptoms: limping present for three months, tremor of the hands for six weeks, falling of the head to the left side for six weeks, bed-wetting for six weeks, pain behind the left eye for three weeks and left earache for several weeks. Lack of concentration and emotional instability were also elicited.

On examination of the patient, ataxia and poor coordination were present; tremor of the left hand and the left lower limb on movement was found. A number of investigations were carried out. At lumbar puncture the cerebro-spinal fluid was under increased pressure; it was clear, and contained no cells; the protein content was 20 milligrammes per 100 mils, the chloride content was 710 milligrammes per 100 mils, and glucose was present. A Mantoux test produced a negative result with a dilution of one in 1000. X-ray examination of the left hip, the spine, the chest and the abdomen gave negative results; the radiographic appearance of the skull suggested slowly developing hydrocephalus. The electroencephalogram was consistent with a subtentorial lesion, but suggested the possibility of a posterior right-sided lesion. Examination of the ocular fundi revealed bilateral papillœdema. Three blood counts gave findings within normal limits.

Posterior decompression was carried out on February 25, and no tumour was found. A ventriculographic examination on March 11 revealed a large cyst, the air being injected into the cyst rather than into the ventricles. The child was operated on, on the same day, and the cyst was removed intact; the fluid contained hooklets and scolices.

During her stay in hospital the patient's condition improved, but she remained emotionally labile up to the time of her discharge on April 1. A Casoni test produced an immediate negative but delayed positive result.

Dr. Nelson said that, as in many cases of cerebral hydatid disease, the diagnosis was not considered at the first examination. The alteration in gait, ataxia and non-coordination suggested a posterior fossa lesion, and it was not until exploration had revealed no abnormality that a ventriculogram showed the true state of affairs. The cyst was punctured instead of the ventricle, and the skiagrams were so typical that immediate operation was carried out.

Ependymoblastoma of the Vermis.

Dr. Nelson next showed a male patient, aged seven years and nine months. He complained of the sudden onset two months previously of nausea and vomiting in the early morning, sometimes during the day and lately in his sleep. For the past two months he had had a headache, which had become much more severe, so that he preferred to lie quietly for most of the time.

On examination of the patient, he was seen to be a red-headed, intelligent, cooperative boy, in no obvious pain, fully conscious and speaking normally. Bilateral papillœdema was present, greater on the left side than on the right, and nystagmus to left and right was observed. The cranial nerves were intact, and diplopia was absent. Coordination was normal, muscle power was unimpaired and the child walked normally. Sensation was unimpaired and the reflexes were normal (both plantar reflexes were flexor in type). An electroencephalogram on March 19 revealed minor abnormalities without localizing signs; the findings suggested an infratentorial rather than a supratentorial tumour. However, there were no cortical foci sufficiently defined to suggest a cerebral lesion. X-ray examination of the skull and long bones revealed no abnormality.

During the child's stay in hospital morning vomiting and the headache continued. He voluntarily limited his activity, though he was allowed up and about the ward. On March 23, 1953, blurred vision was absent from the left eye for a few hours. He had been noticed to be a little unsteady on his feet at times. On March 30 lumbar puncture produced clear cerebro-spinal fluid at a pressure of 105 millimetres of cerebro-spinal fluid; the Queckenstedt test produced a positive result. The fluid contained six lymphocytes per cubic millimetre, and 80 milligrammes of protein per 100 mils. On March 31 vomiting and headache were present, and the child was lethargic.

On April 1, a ventriculographic examination was carried out through a right burr hole, 40 mils of oxygen being introduced. Moderate enlargement of the ventricles was present, and the third ventricle was dilated. It was thought that a posterior fossa block was present and that exploration was necessary.

Posterior decompression was carried out on April 3, and a tumour the size of a large grape was found overlying the foramen of Magendie between the cerebellar tonsils; the tumour was red, soft and friable. The pathologist, Dr. R. D. K. Reye, reported that the specimen was a highly cellular tumour of which the neoplastic cells were fairly uniform in type, being oval to elongated nuclei with cytoplasmic and fibrillary extensions; there were scattered small foci of necrosis, and mitotic figures were fairly numerous. The diagnosis of ependymoblastoma was made.

The patient recovered well, and courses of deep X-ray therapy were commenced on April 21 at the Royal Prince Alfred Hospital. He remained ambulatory in the ward, and had no headache or vomiting.

Dr. Nelson said that the case under discussion was in contrast to the preceding case, as the cerebellar signs were more definite and the patient had a history rather typical of a rapidly growing neoplasm. Ventriculography confirmed the presence of a posterior fossa lesion, and as in most cerebellar lesions, the electroencephalogram revealed no characteristic changes. The child had responded well to deep X-ray therapy, but the ultimate outlook was poor. In spite of that, it was well worth while carrying out an exploratory operation in all cases of posterior fossa lesions, as the astrocytomata had such a favourable outlook.

Cerebral Abscess.

The third patient shown by Dr. Nelson was a boy, aged three and a half years, who had had two attacks at an interval of a few days of headache, fever and anorexia with vomiting on one occasion; the attacks lasted respectively for twenty-four and three hours, and the boy was then well. One week prior to his admission to hospital he had developed a limp in his left leg with a tendency to fall over, but no pain in the leg. Some flaccid paresis was noted in the leg, and Babinski's sign was present; the weakness persisted.

On examination of the patient, consciousness was normal. Bilateral papillœdema was present, greater on the left than on the right. The left abdominal reflexes were absent, the left leg was weak and the knee jerk was absent on the left side; the plantar reflex was extensor on the left side. The patient was afebrile.

An electroencephalogram on March 13 was reported as being a highly abnormal recording; there was a predominant right parieto-occipital δ dysrhythmia consistent with the presence of a cerebral tumour; the diffuse changes suggested raised intracranial pressure. Ventriculography was attempted on March 18. At right ventricular tap thin turbid fluid was obtained which resembled thin pus; 45 mils were removed, and one mil of "Diodrast" was injected, followed by 300,000 units of penicillin. A febrile reaction followed, which lasted for a few days. Intramuscular injections of 300,000 units of penicillin were given every eight hours. Culture of the pus produced no growth of microorganisms. The abscess was aspirated and drained at frequent intervals and further quantities of pus were obtained. Repeated attempts at culture still failed to produce any growth of microorganisms.

On April 26 the child suddenly lapsed into coma with signs of left hemiplegia, and the abscess was again aspirated urgently, with relief of pressure. Open drainage was instituted, and the contents of the cavity were sucked out. "Chloromycetin" and sulphadiazine therapy was instituted. Over the next few days his state of consciousness gradually improved, and the weakness of his left arm and leg also became less pronounced. Drainage from the abscess became less, but frequent dressings and aspirations were continued.

By May 1 the child's consciousness seemed normal, and there was only minimal loss of power in his arm and leg. His condition continued to improve, drainage from the abscess ceased and the scalp wound healed. However, a recent pneumoencephalogram suggested that the abscess cavity was still present; if it persisted it might later need excision.

Dr. Nelson said that the patient had presented clinical signs suggestive of a right-sided cerebral lesion, and that impression was supported by the electroencephalogram. Ventriculography had disclosed a cavity containing thin, turbid fluid, which was unlike the ordinary thick pus found in a cerebral abscess. Those findings, and the subsequent course, suggested that the patient might have a porencephalic cavity which had become infected.

Left Diaphragmatic Hernia.

Dr. Nelson finally showed a male patient, aged four years, who had had intermittent colicky mid-line abdominal pain for eight days, precipitated by food. Vomiting had been present for two days eight days previously, but not since.

On examination, the child was seen to be pale; vague tenderness was elicited round the umbilical area. Numerous exostoses of the ribs were present. There was poor expansion of the left side of the chest, and the apex beat was displaced to the right; increased resonance of the percussion note was present at the base of the left lung, and bowel sounds were heard in the left side of the chest. On July 27, 1952, an X-ray examination revealed the following abnormalities: (i) multiple exostoses, both intrathoracic and extrathoracic, coming from the ribs; (ii) pronounced displacement of the mediastinum to the right; (iii) a large round radio-opaque mass with a crenated margin in the upper part of the abdomen; (iv) bowel extending up the left hemithorax to the clavicles and even across the mid-line to the right.

On August 1, operation was undertaken for repair of the diaphragmatic hernia. The colon was in the left pleural cavity, where many adhesions and some free fluid were present. The left lung was completely atelectatic. A combined thoraco-abdominal approach was used; the colon was returned to the abdomen after the diaphragm had been incised, and the defect was closed. The lax hiatus was tightened, and the pleura and peritoneum were closed. The wound was not drained.

After the operation some fluid and air collected in the left side of the chest, and the patient had a high temperature for a few days, which settled down with penicillin therapy. The fluid was gradually absorbed, and the lung was fully aerated on the patient's discharge from hospital.

Dr. Nelson said that the child's condition was recognized clinically on his first attendance at the hospital. He had presented none of the acute symptoms which were sometimes seen in infancy, and his condition might easily have been dismissed as recurrent appendicitis without a careful clinical examination.

Mesenteric Cysts.

DR. J. STEIGRAD and DR. DAVID L. DEY showed a girl, aged two years and nine months, who had been admitted to the Royal Alexandra Hospital for Children on November 24, 1952, with a small bowel volvulus due to a mesenteric cyst. She was operated on soon after her admission and the volvulus was corrected and the cyst removed. Pathological examination of the specimen showed it to be a large collapsed cyst with a thin translucent wall. The chylous fluid from the cyst contained 61 milligrammes of urea, 110 milligrammes of glucose, 355 milligrammes of chloride, 79 milligrammes of cholesterol and 400 milligrammes of ether-extractable fat, all per 100 millilitres. The wall was not inflamed; it was lined by endothelium, and composed of smooth muscle with a little vascular connective tissue. It appeared to be a chylous mesenteric cyst.

The patient was readmitted to hospital on February 11, 1953, with small bowel obstruction. Operation was performed on February 12, and a segment of small bowel was resected. The pathologist reported that the specimen was a long segment of small bowel with a mesenteric cyst at one point along its length. In that region loops of bowel were adherent by firm fibrous adhesions. In the region of the cyst the bowel took a complete turn upon its axis and pierced the mesentery. At that point the lumen was appreciably narrowed. One of the cysts contained dirty brown material together with material of similar consistency but reddish in colour. Microscopic examination of a section showed it to consist of a portion of small bowel, the subserosa of which was continuous with the wall of a cyst attached to the outer surface of the gut. The mucosa and submucosa of the intestine were congested and œdematous, and macrophages contained blood pigment. The cyst contained blood.

Convalescence was uneventful, and the patient was discharged from hospital on February 26.

Bifid Right Kidney with Double Ureter.

Dr. Steigrad and Dr. Dey also showed a female patient, aged five years, who had been admitted to hospital on March 1, 1953, with a history of dribbling incontinence of urine for ten to twenty minutes after micturition. Micturition was normal in all other respects and there were no urinary or other symptoms. Physical examination of the patient revealed no abnormality. Excretion pyelography revealed normal excretion by both kidneys. There was a bifid right kidney with two ureters. The lower pelvi-calyceal system was drained by a ureter which was of normal size throughout. The upper part of the right kidney was drained by a ureter which was dilated in both abdominal and pelvic spindles and was kinked opposite the third lumbar vertebra. It apparently emptied below the bladder. Both kidneys apparently emptied into the bladder. The left urinary tract and bladder were within normal limits.

On March 12 at operation the upper part of the right kidney and its ectopic ureter were removed. Convalescence was uneventful, and no further urinary dribbling occurred. Microscopic examination of the urine after operation revealed no abnormality. Histological examination of the portion of kidney removed at operation showed it to be the seat of chronic pyelonephritis. The patient was discharged from hospital on March 25, two weeks after operation, and remained well and free from symptoms.

Pre-Sacral Teratoma.

The next patient shown by Dr. Steigrad and Dr. Dey was a female, aged eight years, who had been admitted to hospital on February 18, 1953, with acute retention of urine. The bladder was tense and painful, and displaced to the left. The urethral meatus and vaginal introitus were inflamed. On rectal examination a mass was felt behind the rectum. Penicillin and sulphadiazine therapy was commenced and the bladder was catheterized. The patient had twice been in another hospital for drainage of a cyst behind the bladder in 1946 and 1947. On each occasion she had presented with retention of urine. Excretion pyelography revealed a large soft-tissue rounded tumour extending into the abdomen from the pelvis. Both kidneys were in normal position, and secreted normally. The abdominal portions of the ureters diverged laterally in their lower parts. The pelvic part of the ureters could not be seen. The outline of the bladder could not be identified with certainty, but the dye outlined a large pear-shaped tumour on the left side of the lower part of the abdomen. Old Perthes's disease of the left hip was present. Simultaneous cystography and X-ray examination with a barium enema revealed the bladder and rectum to be displaced anteriorly and to the left.

At operation on March 5 a large multilocular tumour was found attached to the sacrum, pushing up the bladder and uterus and flattening the rectum. Aspiration revealed yellowish fluid containing fat globules, debris and polymorphonuclear leucocytes. The tumour was freed as far as possible from the front and then removed through a posterior incision, with excision of the coccyx. Convalescence was uneventful. Pathological examination showed the tumour to be a large cystic tumour weighng 179 grammes. One portion consisted of a large loculated cyst, the remainder was a mass of bright yellow adipose tissue. On microscopic examination, cartilage and glial tissue were present, and smaller cysts were lined by mucus-secreting columnar epithelium and by squamous epithelium. Smooth muscle was found in the wall of some of the cysts. All the tissues were well differentiated.

The diagnosis was considered to be benign teratoma. The patient remained well, and was discharged from hospital on March 24.

Rheumatism and Hysteria.

Dr. Steigrad and Dr. Dey next showed a boy, aged eleven years, who had been admitted to hospital on February 15, 1953, from the Mater Misericordiæ Hospital, North Sydney, where he had been a patient since January 2, 1953. From September, 1952, he had suffered from recurrent pain in both ears, which became acute in December, 1952, and was associated with a sore throat and fever. This condition settled down after one week's treatment with penicillin. In the latter part of December he developed a painful tender mass, regarded as enlarged lymph nodes, in the right groin, and was again pyrexial. In spite of treatment his condition became worse, the pain extending into his thigh and subsequently down his leg. The pain was made worse by movement. By January he was complaining of pain on micturition. Penicillin was given without response; a subsequent course of aureomycin made him vomit, and was abandoned. Late in the month he was thought to have a fluctuant swelling in the femoral triangle, which was needled, but nothing was found on aspiration. Soon after he had some pain in his back and chest, and some vomiting; but his condition had been largely unchanged until his admission to the Royal Alexandra Hospital for Children. He had had his leg immobilized in a Thomas bed knee splint, but this had afforded him little or no relief. Investigations to that point had included various radiographs of the bones of his thigh, hip and pelvic girdle, all of which appeared within normal limits. The Mantoux test failed to produce a reaction. A blood examination on January 2 gave the following information. The erythrocytes numbered 3,810,000 per cubic millimetre; the leucocytes numbered 11,200 per cubic millimetre, 47% being neutrophile cells, 38% lymphocytes, 9% monocytes, 5% eosinophile cells and 1% basophile cells. On January 13 the leucocytes numbered 19,500 per cubic millimetre, and the erythrocyte sedimentation rate was 29 milli-

metres in one hour (Westergren). On February 9 the erythrocyte sedimentation rate was 18 millimetres in one hour (Westergren). The child had sustained an injury of an obscure nature to his right leg in a motor-car accident some two years before. His mother was a highly strung woman, and the boy also tended to be emotional.

On examination the child appeared superficially healthy, but had a strained and worried expression. There was no visible wasting of either leg, but he moved the right limb only with apparent evidence of much pain. He would not allow any passive movement of joints beyond a few degrees, and was hyperæsthetic over the whole of the thigh, the pubic region, the iliac crest and the buttock, as well as the dorsum of the foot. This hyperæsthesia was not of an anatomical distribution. The right thigh was half an inch greater in diameter than the left in the upper part. Examination of his nervous system gave negative results, palpation of his abdomen revealed no tenderness or mass, and rectal examination revealed no abnormality. Radiographs of his bones and an examination of the cerebro-spinal fluid revealed no abnormality. No infection was detected in his urine. He had a mild pyrexia, and his erythrocyte sedimentation rate (Westergren) was 32 millimetres per hour. A blood count gave the following information: the hæmoglobin value was 14·1 grammes *per centum*; the leucocytes numbered 9620 per cubic millimetre, 56% being neutrophile cells, 32% lymphocytes, 8% monocytes and 4% eosinophile cells.

In view of the absence of any signs but the increased diameter of the thigh in its upper part, surgical exploration of the medial side was undertaken. This revealed no abnormality, and in view of this he was told on recovery from the anæsthetic that he was cured. From that moment on he had full painless movement, and became a cheerful normal boy, in spite of the fact that his temperature rose to 108° F. on the second post-operative day. He remained well. His abnormally high erythrocyte sedimentation rate slowly subsided to 16 millimetres in one hour (Westergren) on April 22. It appeared that his condition was an example of hysterical overlay, the beginning of which was probably a toxic monarticular arthritis.

Erythroblastosis Fœtalis with Perforation of Bowel.

DR. ERIC GOULSTON and DR. C. JOHN GIBSON presented a case of a female infant who had died in the hospital on March 27, 1953, at the age of ten days. The child had been born on March 17, the pregnancy and labour having been normal. The mother was Rh negative. Her first baby had died from an unknown cause. One hour after birth of the present infant, jaundice had been noticed; the hæmoglobin value was found to be 9·4 grammes *per centum*. A Coombs test produced a positive result; the liver and spleen were found to be enlarged. An immediate replacement transfusion was given, and treatment was started with penicillin and streptomycin. On March 18 the jaundice had slightly increased, and abdominal distension was noticed. Vomiting began and persisted for two days. On March 19 the child was transferred to the Royal Alexandra Hospital for Children. The jaundice was increasing, abdominal distension was present, and bowel sounds were not audible, but no fluid was found in the adomen. Plain X-ray examination of the abdomen showed gas-distended loops of ileum, in one of which there was a fluid level. Free gas was present in the peritoneal cavity. No gas was seen in the colon. The X ray indicated, firstly, perforation of the gut and, secondly, a probably coexistent small bowel obstruction. The platelet count was 50,000 per cubic millimetre of blood, and the hæmoglobin value was 16 grammes *per centum*. On March 20 another plain X-ray examination of the abdomen showed that small bowel obstruction was still present. There was an increase in gaseous distension of the bowel and an increase in free gas in the peritoneal cavity since the previous examination. At laparotomy, the small bowel was found to be hæmorrhagic and friable, there were multiple perforations throughout the bowel, and peritoneal fluid present was meconium-stained. The large bowel appeared normal. The child's condition improved slightly for the first few days post-operatively and then gradually deteriorated, ending in death. At autopsy, the main findings were: (i) signs of erythroblastosis—enlarged liver and spleen, generalized œdema; (ii) mesenteric venous thrombosis; (iii) multiple perforations of gangrenous small bowel; (iv) peritonitis.

Imperforate Anus with Perforation of Bowel.

Dr. Goulston and Dr. Gibson next presented the case of a baby who had been born on October 26, 1951, with a birth weight of seven pounds one ounce after normal pregnancy and labour. On October 29 the baby vomited and passed meconium-stained urine. It was then referred to the Royal Alexandra Hospital for Children and admitted. It was found to be a cyanosed baby with a faint cry. The abdomen was distended, and no bowel sounds were audible. A meconium stain was found on the napkin. No anus was present, but there was a suggestion of a dimple in the perineum, which bulged when the infant cried. Plain X-ray examination of the abdomen revealed the presence of a large pneumoperitoneum. The distal segment of bowel was seen to lie about an inch from the anus. The baby died on the same day as it was born. At autopsy it was found that the lungs were only partially aerated—namely, at the anterior aspects of the lobes; the remainder of the lungs was atelectatic. No areas of pneumonic consolidation were demonstrated. A large amount of meconium was present in the peritoneal cavity with resulting meconium peritonitis. A perforation was demonstrated in the sigmoid colon. The alimentary tract distal to the perforation was normal in position and fixation. Distally, the pelvic colon ended at the bladder neck. When the colon was opened, the lower end was found to be funnel-shaped, its narrow extremity communicating with the prostatic part of the urethra. The urethra was otherwise normal, as were the kidneys, ureters and bladder. The final diagnosis was: (i) partial atelectasis (bilateral), (ii) imperforate anus, (iii) ruptured sigmoid colon with meconium peritonitis, (iv) recto-urethral fistula.

Hirschsprung's Disease with Perforation of Bowel.

Dr. Goulston and Dr. Gibson next presented a baby, aged five and a half months, who had been admitted to hospital in January, 1953, at the age of two days. The child had been born on January 12 with a birth weight of eight pounds after a normal pregnancy and labour. On January 13 the baby's condition had been satisfactory and the breast was being taken. One meconium-stained stool was passed. On January 14 a grunting cry developed, with slight cyanosis and muscular twitching. The abdomen was found to be greatly distended with gas, and bowel sounds were absent, but there was no vomiting. The baby was referred to the Royal Alexandra Hospital for Children, where the presence of jaundice was noted. The abdomen was very distended, with prominent veins visible, and was tympanitic on percussion. Bowel sounds were very infrequent, but no masses were palpable and the umbilicus was dry and clean. Plain X-ray examination of the chest revealed no abnormality, but in the abdomen a considerable amount of free fluid was present in the peritoneal cavity. Some gas-distended loops of small intestine were visible, and the transverse colon was seen to be distended with gas. A loop of colon was seen in the lateral view to contain solid material mixed with small bubbles of gas. It was considered that the loop might be ascending or descending colon, probably the former. The radiologist commented that such an appearance had been attributed by Neuhauser to inspissated meconium found in cases of meconium ileus. The X-ray evidence indicated (i) perforation of a gas-containing abdominal viscus into the peritoneal cavity and (ii) intestinal obstruction, probably at the splenic flexure. The probable cause of this was considered to be pancreatic insufficiency. At laparotomy a spastic segment of bowel was seen at the splenic flexure, where the bowel was ruptured, causing meconium peritonitis. The perforation was oversewn, and a transverse colostomy was performed. The baby's subsequent progress had been uneventful. It was quiet, contented, thriving, and gaining in weight. Hemicolectomy was to be performed in another six to eight weeks.

Tracheo-Œsophageal Fistula.

Dr. Goulston and Dr. Gibson presented three babies who had undergone surgical treatment for tracheo-œsophageal fistula. The first baby had been born on April 21, 1953, after a normal pregnancy and labour. It had breathed poorly at first, but it had revived fairly quickly. On the day after birth the baby had vomited thick brown mucus and was regurgitating all food immediately. The next day it was still vomiting and tube feeding was attempted, but the tube could not be passed into the stomach. On April 25 the baby was transferred to the Royal Alexandra Hospital for Children. X-ray examination with lipiodol showed that the upper part of the œsophagus ended blindly at the lower border of the second thoracic vertebra. A fistula connected the lower part of the œsophagus with the tracheo-bronchial tree, probably in the region of the carina. Thoracotomy was carried out, the fistula was divided and sutured, and end-to-end anastomosis of the œsophagus was performed. On May 2 fluids were given by mouth, but on May 6 the child was vomiting every feed and gastrostomy was performed. On May 21 the child was progressing well. It was considered that œsophageal dilatation would be needed in the future.

The second child had been born on April 13 after a normal pregnancy and labour. On delivery the baby was very "mucousy" and slightly cyanosed. The throat had to be sucked out frequently in the next few days. The baby suffered from recurrent attacks of cyanosis, which were relieved by the sucking out of the throat. When bottle-feeding was commenced the baby became distressed and cyanosed and regurgitated the whole feeding. On April 16, it was transferred to the Royal Alexandra Hospital for Children. X-ray examination with lipiodol revealed that the œsophagus appeared to end in a blind pouch at about the level of the third thoracic vertebra. On April 17 thoracotomy was carried out with end-to-end anastomosis of the œsophagus. On April 23 the oral administration of fluids was commenced, but on April 29 the baby was still vomiting frequently. On April 30 X-ray examination with lipiodol revealed an œsophageal stricture, and gastrostomy was performed. On May 13 chest X-ray examination showed patchy consolidation in both lungs. By May 21 good progress had been made and the X-ray appearances of the chest were normal. It was considered that the child would require œsophageal dilatation in the future.

The third baby had been born on January 13 after a normal pregnancy and labour. It had sucked poorly and vomiting had followed immediately. Tube-feeding was attempted, but obstruction to the tube was met with four and three-quarter inches from the mouth. On January 14 the child was transferred to the Royal Alexandra Hospital for Children, where examination revealed that mucus was collecting in large amounts in the mouth; this necessitated frequent suction and continuous intranasal administration of oxygen. X-ray examination with lipiodol revealed that the proximal part of the œsophagus had been outlined with lipiodol; it was represented by a blind pouch lying opposite the second thoracic vertebra. An attempt to catheterize the fistula between the bronchial tree and the lower œsophageal segment was unsuccessful. At thoracotomy a tracheo-œsophageal fistula was found. The fistula was closed, and end-to-end anastomosis of the œsophagus was performed. On January 20 gastrostomy was performed. On February 23 oral feeding was tried. There were no ill effects for several days, then the child became cyanosed. On March 31 œsophagoscopy and œsophageal dilatation were commenced. Since then the child had been having weekly dilatations of the œsophagus and was being fed completely through the gastrostomy tube. On April 16 oral feeding was again commenced, and by May 2 the child was taking all feeds by mouth with no difficulty, vomiting or cyanosis. It was being fed through the gastrostomy tube only for twenty-four hours after œsophageal dilatation. X-ray examination with a barium bolus revealed that an œsophageal stricture was still present. By May 22 good progress had been made.

Adenocarcinoma of the Testis.

The last patient shown by Dr. Goulston and Dr. Gibson was a boy, aged four years, who had been admitted to hospital in February, 1950, at the age of seven months. One month previously the mother had noticed that the child's left testis was larger than the right, and it had been increasing in size steadily since; it was not painful. Examination of the child revealed a large tumour in the left side of the scrotum, from which the testis could not be freed. The tumour was hard, free from the skin and deep structures, attached to the cord and not translucent. The right testis felt normal. No inguinal glands or abdominal tumour could be felt. On February 28, 1950, left orchidectomy was carried out. The tumour appeared to involve the whole of the testis, the latter being surrounded by a small secondary hydrocele. Histological examination revealed that the tumour was an embryonal adenocarcinoma of high-grade malignancy. A course of deep X-ray therapy was given to the whole glandular chain in the thorax and abdomen, ten treatments being given. On March 30, 1950, the child was readmitted to hospital and given a further course of ten treatments of deep X-ray therapy. The child had been examined every three months since that time and appeared to be perfectly well.

Bladder Neck Obstruction, Bilateral Hydronephrosis and Hydroureters.

DR. E. S. STUCKEY showed a boy, aged four years, who had been admitted to hospital on July 19, 1952, with a history of chronic urinary difficulty since birth, frequent enuresis, frequency of micturition and passing of small amounts of urine only, the stream being dribbling and thin. Physical examination revealed only a large palpable bladder. Investigations showed a blood urea content of 47 milligrammes per centum, poor urea clearance and sterile pyuria. There was no dye seen after two hours with excretion pyelography. Cystoscopy was performed after urethral dilatation without difficulty and showed a large bladder with trabeculation and funnel neck; the ureteric orifices were not visible. At operation the bladder wall was found to be grossly thickened, and in view of the small funnel bladder neck, wedge resection of the bladder neck by diathermy was performed with cystostomy. Passage of sounds and repeated catheterization were required after operation, and the cystostomy wound continued to drain copious urine. The child could pass urine only with straining and still with dribbling of only small amounts. In October, 1952, excretion pyelography after seven hours showed good dilatation of both ureters, pelves and calyces. Although urethrography in November, 1952, showed no urethral obstruction, further wedge resection and suprapubic cystostomy was performed on November 26, as several posterior urethral folds were found to obstruct the passage of urethral sounds out of the bladder. Despite persistent pyuria and the need for frequent dilatation and catheterization, the cystostomy wound gradually healed, and by February, 1953, the child was passing urine *per urethram* without difficulty in larger amounts and with a good stream. In April, 1953, excretion pyelography still showed gross bilateral hydronephrosis and hydroureter, but cystography showed no ureteric reflux and less bladder trabeculation, and excretory urethrography showed a wide funnel neck of bladder with no evidence of obstruction.

Dr. Stuckey said that the child still had occasional attacks of pyuria, and had had two attacks of right epididymo-orchitis after instrumentation, the last in May, 1953, requiring incision and drainage of the scrotum. He now had almost complete control and passed urine every two hours during the day by request, with no straining and with a good stream.

Bilateral Stenosis of Pelvi-Ureteral Junctions with Hydronephrosis.

Dr. Stuckey then showed a girl, aged fifteen months, who had been admitted to hospital on May 19, 1952, at the age of eight weeks with a history of feeding difficulties, fainting turns and a large cystic mass in the right hypochondrium, which was gradually increasing in size. Retrograde and intravenous pyelography showed the lower end of the right ureter dilated and tortuous, but the pelvis and calyces were not visualized. There was some dilatation of the left renal pelvis and major calyces with dilatation and tortuosities of the left ureter. At operation on September 28, 1952, marked stenosis at the right pelvi-ureteral junctions was seen; this area was excised, and nephroplasty and nephrostomy were performed. Because of continued pyuria and a second nephrostomy draining 16 ounces of purulent urine, right retrograde and subcutaneous pyelography was performed, which showed complete block at the right pelvi-ureteral junction, and no dye in the right pelvis. Almost complete absence of function in the right kidney was confirmed by a very poor urea clearance test result, although urea clearance from the left kidney was normal. On September 8, 1952, the left kidney was explored and a large kidney was found with intrarenal hydronephrosis and a pelvi-ureteral junction small in relation to the ureter, although patent. This junction was incised longitudinally and then reconstituted over a splinting ureteric catheter with nephrostomy. Right nephrostomy was performed three weeks later, and marked hydronephrosis and chronic pyelonephritis were found, with complete congenital stenosis of the right ureter and pelvi-ureteral junction. Cystography two weeks later showed no left ureteric reflux and a smooth bladder outline. A urea clearance test on November 13, 1952, showed 2·2% concentration of urea in three hours.

Dr. Stuckey said that the child had had persistent mild pyuria since discharge from hospital on November 19, 1952, but excretion pyelography on February 27, 1953, showed that the left hydronephrosis was much diminished, although there was still some dilatation of the pelvis and major calyces, and the ureter was kinked at the level of the iliac crest and somewhat dilated. The child now passed a normal amount of urine without difficulty and seemed to hold urine for at least one hour.

Right Hydronephrosis.

Dr. Stuckey's next patient, a baby girl, had been admitted to hospital on September 19, 1952, with a history of difficult feedings since birth and repeated vomiting attacks, usually in association with the passing of offensive urine, fever and vulval excoriation. She had always had complete incontinence of urine with continual dribbling of small amounts. Investigations showed no physical abnormalities, a blood

urea content of 27 milligrammes *per centum*, and pyuria with profuse growth of *Bacterium coli* in a catheter specimen of urine. Excretion pyelography showed well-marked, generalized hydronephrosis of the right kidney with a normal left urinary tract. Cystoscopy showed a large atonic bladder, trigonitis and an œdematous and inflamed ureteric orifice. Right retrograde pyelography confirmed the hydronephrosis, but showed no definite evidence of pelvi-ureteral block. At operation on May 5, 1953, through a right lumbar incision, a large hydronephrotic kidney was seen with a grossly dilated renal pelvis and stenosis at the pelvi-ureteral junction. The stenotic site was incised longitudinally and then reconstituted over a ureteric catheter splint, and a nephrostomy was performed. The child made an uninterrupted recovery with good drainage from the nephrostomy tube, the urine of which remained sterile with aureomycin. A catheter specimen of urine taken two weeks after operation was also sterile.

Dr. Stuckey said that she had had no post-operative vomiting, but was still incontinent at times, although she was passing larger amounts of urine at less frequent intervals. Excretion pyelography three weeks after operation had shown no substantial decrease as yet in the hydronephrosis.

Bullet Wound to Right Ureter.

Dr. Stuckey's last patient, a boy, aged twelve years, had been admitted to hospital on April 25, 1952, several hours after being shot by a 0·22 bullet, which had entered his right loin and emerged just to the right of the umbilicus. Operation through a right pararectal incision showed generalized peritonitis, with perforation of the third and second parts of the duodenum, the jejunum and the transverse colon. The perforations were oversewn. There was a right fibro-peritoneal hæmatoma around and below the right kidney, and the hole of entrance to the peritoneal cavities was oversewn and the peritoneal cavity drained suprapubically. By May 5, 1952, there were signs of an abscess in the pelvis or right loin, and incision of the old bullet entrance wound in the right loin produced a large amount of thin blood-stained fluid. Intravenous indigo carmine was excreted from that site one hour after injection. At the second operation on May 19, 1952, cystoscopy and right ureteric catheterization were performed, and the right kidney and ureter were exposed by a right lumbar incision. By injecting indigo carmine up the ureteric catheter, the site of ureteric puncture, two inches from the pelvi-ureteral junction, was found, and the ureter was reconstituted over the ureteric catheter. Excretion pyelography on May 29, 1952, showed fairly well-marked right hydronephrosis with a narrowing of the right ureter at the site of injury at the upper border of the fourth lumbar vertebra. However, excretion pyelography on January 19, 1953, showed no evidence of hydronephrosis of the right pelvi-calyceal system, although there was a mild degree of urinary stenosis proximal to the point of injury in the ureter, with slight dilatation of the ureter, half an inch distal to that point.

Recurrent Dislocation of the Patella.

DR. M. SOFER SCHREIBER showed a girl, aged six years, who had fallen over whilst skipping in May, 1952. Her left leg was stiff, and she limped for a few days. Subsequently she spared her knee when running, but did not complain of discomfort. Her parents noted that her knee was an odd shape. On examination of the child in April, 1952, wasting of the left quadriceps muscle was evident, and there was a full range of painless movement. The patella, which seemed normal in size and shape, was dislocated laterally at about 30° of flexion. No significant *genu valgum* was present. X-ray examination showed no abnormality other than lateral dislocation of the patella where the knee flexed. Physiotherapy to build up the quadriceps muscle did not alter the condition. At operation on September 27 the lateral half of the *ligamentum patellæ* was detached from its insertion into the tibial tuberosity, and was passed medially beneath the medial half. It was then sutured firmly to the periosteum and aponeurotic expansions on the medial aspect of the upper end of the tibia (Goldthwait's operation). The leg was subsequently immobilized in plaster for six weeks. When the child was allowed to flex her knee again, it was found that the patella still became dislocated laterally on 30° of flexion. Accordingly a more radical operation was performed on February 6, 1953. Through a long lateral incision the quadriceps was realigned by longitudinal division of the ilio-tibial band, *vastus lateralis* muscle and lateral capsule of the knee joint. The tibial tuberosity was completely detached, and was reimplanted on the medial aspect of the upper end of the tibia.

Cardio-Œsophageal Relaxation Simulating Pyloric Stenosis.

Dr. Sofer Schreiber's second patient was a boy, an only child, who had been admitted to hospital in December, 1952, at the age of ten weeks, with a provisional diagnosis of pyloric stenosis. He had been vomiting for nine weeks; the vomiting was projectile and the vomitus was occasionally brown in colour. The birth weight had been seven pounds four ounces. His weight on admission to hospital was six pounds twelve ounces. Very well-marked peristaltic waves were observed to pass across his epigastrium. A diagnosis of pyloric stenosis was made, and laparotomy was performed on that day. The pylorus was found to be thicker than usual, but no pyloric tumour was present. A provisional diagnosis of pylorospasm was made. However, vomiting continued, and the vomitus was occasionally brown. His weight was seven pounds four ounces (his birth weight) on January 9, 1953, when he was fourteen weeks old. A provisional diagnosis of hiatus hernia was made, and a barium meal was given. The pylorus opened one minute after ingestion of the meal. Its length (0·3 centimetre) was within normal limits. Peristaltic waves passed normally across it. Free regurgitation of fundal contents into the œsophagus occurred with every respiration. No hiatus hernia could be demonstrated. The stomach emptied three hours after ingestion of the meal. A final diagnosis of cardio-œsophageal relaxation was made, and the child was nursed sitting upright. The vomiting ceased, and a satisfactory weight gain commenced. Dr. Sofer Schreiber made the comment that the diagnosis made should be considered in the differential diagnosis of pyloric stenosis, especially if any hæmatemesis or brown vomitus was reported. The palpation of a pyloric tumour should differentiate the condition, but such a tumour could not be felt in every case of pyloric stenosis.

Barium meal examination in doubtful cases would differentiate pyloric stenosis from cardio-œsophageal relaxation and hiatus hernia. The symptoms in the present case were thought to be due to œsophagitis caused by the reflux, and to reflux pylorospasm.

Chylous Cyst of the Mesentery.

DR. D. COHEN showed a boy, aged three years, who had been admitted to hospital with a history of persistent colicky central abdominal pain and vomiting of two days' duration. About six months previously he had had a similar attack which had subsided. He always tended to be constipated and prior to admission to hospital had been given an enema, which he had retained. On examination shortly after admission to hospital he did not seem to be in distress. There was some deep tenderness in the epigastrium and right iliac fossa. There was no guarding, and no masses were palpable. No abdominal distension was noted. A plain X-ray examination showed multiple fluid levels, but these were considered to be due to the retained enema. This was passed during the night, and a further X-ray examination the next morning showed no abnormality, as did a barium enema examination. The child continued to have recurrent bouts of colicky pain without any vomiting, so it was decided to perform appendicectomy and exploration of the terminal part of the ileum. The abdomen was opened through a McBurney's incision, and immediately a large soft tumour presented. The incision was enlarged by cutting across the right rectus, and the tumour was delivered. It was soft and cystic and contained chylous fluid. It lay between the layers of the mesentery of the terminal part of the ileum and was approximately the size of an infant's skull. It was necessary to remove about a foot of the terminal part of the ileum with the tumour, bowel continuity being restored by end-to-end anastomosis. Convalescence was uncomplicated, the child being discharged on the tenth post-operative day. Pathological examination confirmed that the tumour was a chylous cyst of the mesentery.

Subsymphyseal Exstrophy of the Bladder.

Dr. Cohen's last patient, a girl, aged one year, had been noticed at birth to have an abnormality of the external genitals. On examination she was found to have a bifid clitoris with a band of tissue stretching from behind the anus to the *mons veneris* between the two halves of the clitoris. There was an umbilical hernia, and the umbilicus was placed lower than usual. The abdominal wall below the umbilicus was atrophic. With the child under general anæsthesia, the band of tissue mentioned was divided. This revealed an anus posteriorly in an ectopic position, displaced about one inch anterior to the normal site. The perineal body was deficient. The vagina appeared normal. The urethra was wide and patulous and appeared incompetent.

urine trickling from the bladder with suprapubic pressure. Nothing further was done at that stage, and since then the child had developed normally in every respect. At one year of age she was dry for up to two hours and seemed to be developing satisfactory control. It was therefore proposed to treat her conservatively for the present until it was seen how efficiently bladder control developed.

Horseshoe Kidney.

Dr. Cohen's next patient, a girl, aged ten years, had been suffering from recurrent attacks of left-sided abdominal pain over the last two years. A letter from her doctor stated that she had been examined in the last attack and, clinically, the condition was like a renal colic. On examination, she was a tall, thin girl of nervous temperament. No abnormality was revealed by clinical examination or by microscopic examination of the urine. Excretion pyelography showed good kidney function on both sides. There was a minor degree of dilatation of the left renal pelvis, but no loss of cupping of the calyces. Both kidneys were rotated, and, in oblique views, there seemed to be some anterior displacement of the upper third of both ureters. The appearances were suggestive of horseshoe kidney. Pre-sacral perirenal pneumography was performed with Blackwood's technique. Four hundred mils of air were injected on each side. This confirmed the diagnosis of horseshoe kidney; the bar of tissue across the mid-line uniting the lower poles of the two kidneys was clearly seen. Dr. Cohen said that the patient had been kept under observation since and had continued to have some pain, but, as there was no evidence of any obstruction or urinary infection, no operative interference would seem to be warranted at the present stage.

Delayed Ossification.

Tarsal Bones.

DR. GORDON COLVIN first showed a child who had been brought to the hospital at the age of one year and eight months, suffering from gross plano-valgus deformity of both feet. Crooked and elongated heels were ordered, and the child was to wear boots. No improvement had occurred in seven months, when the feet were radiologically examined for possible bony deformities. The skiagrams revealed rudimentary calcanei and tali and poor ossification of the cuboid.

Deformity of the feet was still present fifteen months after the child had begun to wear boots with altered heels. At this time outside irons and inside T-straps were ordered, together with a wedge one-sixth of an inch inside to each heel. Two months later, both feet were moulded and plaster casts were applied. The casts were worn for eight weeks, with considerable improvement in the appearance of the feet. After their removal the child reverted to the use of boots, irons and T-straps. Skiagrams four months later revealed improvement in the condition of the talus and the *os calcis*, and the navicular was fairly well formed. Eleven months later, when the child was aged four years and eight months, there was still some valgoid roll-over without support. Skiagrams revealed further improvement in tarsal bone formation.

Dr. Colvin said that the patient would require to wear boots with irons and T-straps until the condition was fully controlled.

Upper Femoral Epiphyses.

Dr. Colvin's second patient had been referred from a private hospital at the age of five weeks with the suggestion of a hip abnormality. A skiagram on July 28, 1952, revealed well-formed acetabula with an apparent widening of the joint space and no evidence of epiphyses for the femoral heads.

The patient was treated with the legs in wide abduction by the use of a thick mass of napkins between the legs. Pin-point epiphyses were seen in the next skiagram taken on August 28, 1952. Abduction of the legs was continued. Skiagrams taken on December 31, 1952, showed that the epiphyses were now well developed. Clinically the hips felt normal. Abduction was continued until a skiagram taken on March 16, 1953, showed epiphyses of normal appearance. The child had now commenced to stand, at the age of nine months.

Gross Bony Deformities due to Malnutrition.

Dr. Colvin next showed an aboriginal child, aged six years and nine months, who had been referred by the Child Welfare Department. Previously he had been wandering the bush with his father in an apparent state of gross neglect. He was admitted to hospital on June 25, 1951. Gross skeletal deformities were present, particularly of the lower limbs; both tibiæ were sabre-like in appearance.

A number of investigations were undertaken. The Wassermann and Kahn tests failed to produce reactions. A blood count gave the following information: the hæmoglobin value was 12·7 grammes *per centum*; the red cells were normocytic and normochromic; the leucocytes numbered 15,000 per cubic millimetre, 61% being neutrophile cells, 31% lymphocytes, 3% monocytes and 1% basophile cells. The serum calcium content was 9·4 milligrammes *per centum*, the serum phosphorus content 4·9 milligrammes *per centum*, the serum alkaline phosphatase content 35 King-Armstrong units per 100 millilitres (normal for this age being up to 25 King-Armstrong units per 100 millilitres), and the blood urea content was 27 milligrammes *per centum*.

Skiagrams were reported on as showing pronounced bending of the long bones. The main change occurred about two or three inches from the joints. The calcium content of the bones was good and the epiphyses appeared normal. The whole appearance suggested that an osteomalacic condition had occurred several years earlier and was now healed. The skull appeared normal.

On July 19 osteotomy of the right tibia was performed; the limb was manipulated and put in plaster again on July 26.

On September 27 osteotomy of the left tibia was undertaken.

On November 10, 1952, there was complete union at the osteotomy sites, and good tibial alignment was present.

On May 18, 1953, the alignment of the tibiæ was greatly improved, although there was gross external rotation of the limbs, particularly the right. Despite this and a pronounced femoral deformity, the child walked well with the feet in correct alignment. Femoral osteotomy, previously considered necessary, was postponed indefinitely.

Dr. Colvin said that it was considered that the case showed the results of gross malnutrition at an early age. The beneficial effects of osteotomy in correcting deformities were well illustrated.

Tumour of the Left Calf.

Dr. Colvin finally showed a child, aged one and a half years, who had presented with an enlarged left calf. This had been noticed by the mother only one week before. On examination of the patient, there was a softish, well-defined mass in the left mid-calf. X-ray examination of the left tibia and knee revealed an ovoid mass the size of a small hen's egg, very suggestive of a lipoma.

At operation on May 15, 1953, two months after the first examination of the patient, the tumour was found located within the soleus muscle and "shelled out". The fibres of the soleus in the region were stretched and thinned out by the tumour.

Macroscopic examination of the specimen showed it to be a yellow, completely encapsulated tumour weighing 33 grammes and measuring 4·5 by 4·3 centimetres. The cut surface was uniform and consisted of lobules of pale yellow fat. Microscopic examination showed the tumour to be composed of lobules of adult adipose tissue, which had developed in and about striated muscle, the fibres of which were seen in small groups in its substance and about the interlobular trabeculæ as well as at the periphery. The diagnosis of lipoma was made.

Dr. Colvin said that the patient was at present wearing a plaster cast to prevent a possible equinus deformity due to contracture of the calf muscles while healing was taking place; the cast would be discarded in six weeks.

Gradenigo's Syndrome.

DR. NORMAN MEACLE showed a girl, aged four and a half years, who had been admitted to hospital on February 25, 1953. She had had three attacks of left middle ear disease in the preceding nine months, the last attack four months before her admission to hospital. The symptoms were earache and fever, but she had no discharge from the ear and no deafness. She had been given penicillin on two occasions and had been quite well between attacks. One week before her admission to hospital she had complained of a sore left ear and was feverish. There was no discharge from the ear. She was given sulphonamides for two days, but the pain and fever persisted and she seemed very ill; so she was given aureomycin, one half-capsule four times a day, up to the time of her admission to hospital. With the present attack she was rather deaf and wanted the blinds kept down, and she also complained of sore eyes. There was

no redness or discharge from the eyes. On the day of her admission to hospital she had a small amount of blood-stained, yellow discharge from the left nostril, and her parents noticed that her left eye was turned in. She complained of double vision and kept her left eye covered. She had been drowsy for several days, but was mentally alert. She had had no rigors or high fever. When she was admitted to hospital her temperature was found to be normal and her pulse rate 112 per minute. Examination revealed the presence of left otitis media and left lateral rectus palsy. The left trigeminal nerve was not involved, and there was no fever, papilloedema, neck stiffness or mastoid tenderness. Left myringotomy was performed with the production of sero-sanguineous fluid from which *Streptococcus viridans* was grown; it was sensitive to all antibiotics. X-ray examination of the mastoid and petrous bones revealed no abnormality. Penicillin therapy was commenced. On the following day the physical signs were unchanged, although the temperature was 100° F., and mastoidectomy (Schwartze type) was performed. A cellular mastoid was found with considerable necrosis involving zygomatic cells. The dura mater of the middle fossa was exposed, and granulations were found. The lateral sinus was not exposed. No abnormality was found in cerebro-spinal fluid obtained by lumbar puncture. The administration of sulphadiazine was commenced, and the penicillin therapy was continued. On February 28 the squint was still present, but the temperature was normal and there was no drainage from the wound. By March 2 the squint had disappeared, the temperature had not been above 99° F. and penicillin therapy was suspended. On March 9 the stitches were removed, the wound was clear and the ear was dry. The honorary ophthalmologist reported the absence of any eye lesion. On March 13 the temperature rose to 103° F. for no obvious cause. Sulphadiazine therapy was recommenced, and by the following day the temperature was normal. Within twenty-four hours it had again risen to 105° F., although the child said that she had no pain anywhere. She had constant lateral nystagmus on looking to either side, and although she could sit up without feeling giddy, she became giddy on standing, and refused to walk. She had no intention tremor or dysdiadochokinesis, and the ocular fundi were normal. Treatment was commenced with chloramphenicol. By the next day the temperature had fallen to 99·4° F., but the nystagmus was still noticeable and the patient felt wobbly when she stood. Ocular movements were full, and there was no diplopia or abnormality of the fundi. The mastoid was reopened and the lateral sinus exposed, but the mastoid cavity was found to be clean and no peridural abscess was detected. A blood count revealed a moderate degree of leucocytosis. By March 19 the temperature was still raised, but the nystagmus had disappeared. On March 21 the child complained of difficulty in chewing, but no abnormality was found; and on March 24 she was free from complaints, and her temperature was normal. Subsequent convalescence was without significant abnormality. On May 25 adenoidectomy was performed.

Deafness in Babies.

DR. PATRICIA DAVEY showed three babies to illustrate the diagnosis and treatment of deafness in infancy. The first patient, a girl, had been first examined in December, 1952. Her mother had had no illness during pregnancy. The child had suffered from measles with *otitis media* at the age of five weeks. When examined, she did not turn her head to sound, but responded to loud bangs. It was found that she was not localizing the source of loud sound. Her hearing loss was probably 40 to 50 decibels above threshold. By April, 1953, training had brought about improvement of auditory attention, and hearing now appeared more acute in the left than in the right ear. Dr. Davey pointed out that auditory training at that stage was very important. Even if hearing remained stationary, acuity would improve and speech would start at the normal time.

The next patient, a boy, had been found to have anotia at birth. The external ear was malformed, and the external auditory meatus was atretic on both sides. The mother was given instructions to bring everyday sounds and voices close to the baby's ears. On the assumption that the inner ear mechanism was normal, it was considered that the factor of distance elimination should compensate for the middle ear deafness. So far, training tended to show that that was so. The baby definitely listened with his left ear. Dr. Davey said that the procedure would probably involve continued auditory training, soon with a hearing aid. Plastic operation on the external auditory meatus would not be considered for some years.

The third patient, a boy, had been under treatment for congenital cataract and heart disease, and his parents had noticed deafness at the age of two or three months. His mother had suffered from rubella during pregnancy. At the time when deafness had been first noted auditory training was instituted. It was found that localization of sound was difficult for the baby, and the hearing appeared to be at a level of 40 to 50 decibels at the meatus. Dr. Davey said that the problem was an educational one. With continued intensive auditory training the visual problem would probably prove the greater, and the child would be best placed in a school for the blind.

Commenting on the whole question, Dr. Davey said that suspected deafness in a baby demanded investigation from the earliest possible moment. No baby was too young to be tested or to be prepared for testing. The advantage gained by every extra week of home training might make all the difference between a socially acceptable, talking individual and a finger-spelling introvert, unable to understand or to make himself understood by his fellow men. Auditory training sharpened hearing acuity. Watching the mother's face laid the foundations of lip-reading, which grew automatically. Feelings of social security were automatically laid. Behaviour problems did not occur, and false impressions of mental retardation did not arise. Successful practice along the lines laid down would lay the foundations for the trained teacher of the deaf to give a normal school curriculum to a pleasantly speaking child, however slight the residual hearing.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE SENDING OF QUININE SULPHATE TO PORT ESSINGTON.[1]

Colonial Secretary's Office,

April 1, 1844.

The Deputy Inspector-General of Hospitals.

Sir,

I am directed by His Excellency the Governor to suggest to you the propriety of sending a supply of sulphate of quinine to Port Essington by Mr. Earle who will sail in the "Bintang" for that settlement in the course of the present week. The extent to which intermittent fever is known to have lately prevailed at the settlement (though it was previously unknown there) leads His Excellency to apprehend that they may be in want of this valuable medicine.

I have, etc.,

W. ELYARD.

Obituary.

EDMUND BRITTEN JONES.

We are indebted to Dr. Leonard Lindon for the following appreciation of the late Sir Edmund Britten Jones.

By the death of Edmund Britten Jones on September 30, 1953, the community of South Australia, and the medical profession in particular, have suffered a great loss. It is extremely difficult to express in words an account of the influence exerted upon his fellow practitioners by his ability, his character, and his humanity; and this can only be regarded as an inadequate record of his life and achivements.

Edmund Britten Jones was born on October 8, 1888, and his early education was obtained at Christian Brothers' College, Adelaide. He entered the medical school in 1906, and completed a brilliant course by gaining the Everard Scholarship, having been placed top of the first class honours in his final year. Indeed, he was one of that small number of medical students who, since the inception of the medical school, have achieved first class honours in every year of the course. As a Rhodes Scholar he went up to Magdalen College, Oxford, in October, 1912, and in 1914 had obtained a first in the honours school of physiology. At the outbreak

[1] From the original in the Mitchell Library, Sydney.

of the first war he joined the Royal Army Medical Corps and served as a captain in the 78th Field Ambulance from 1915 to 1919.

Returning to South Australia in 1919, he conducted general practice at Penola, and then at Henley Beach. He returned to England in 1926, proceeded to the degree of M.A. (Oxon.) and the diploma of M.R.C.P. (London) and studied also in Paris and Vienna. From this time onwards he practised as a consulting physician in Adelaide, serving as honorary physician to the Children's Hospital from 1929 to 1947, and from then as honorary consulting physician to that hospital. In 1938 he became one of the Foundation Fellows and a Councillor of The Royal Australasian College of Physicians, and in 1942 was elected a Fellow of the Royal College of Physicians of London. With the outbreak of the second World War he enlisted in the Australian Army Medical Corps; and he served in the Middle East with the rank of lieutenant-colonel in 1940-1941, and was mentioned in dispatches. He was a member of the Council of the University of Adelaide from 1944 until his death. His great record of service and leadership was recognized by the honour of knighthood in January, 1953.

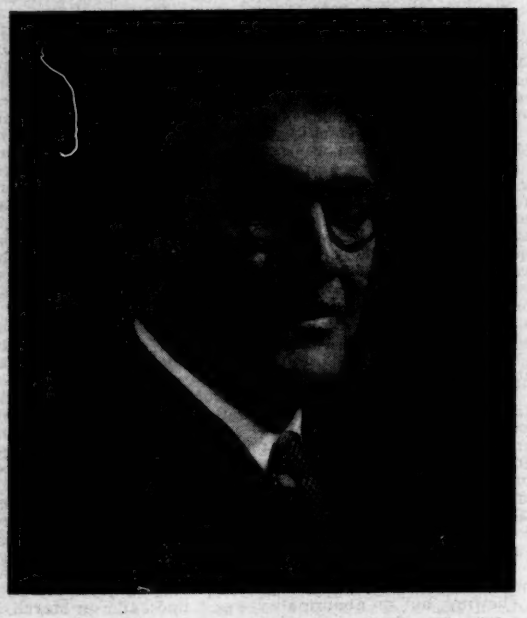
In the practice of his profession Britten Jones owed much to his phenomenal memory; but, even more important, he had the "divine spark" of imagination, and his diagnostic hunches had a habit of being correct. To these qualities were added great judgement and common sense, and an up-to-date knowledge of treatment. Thus it was inevitable that he should have become a really first-class and overworked consultant. His services were sought and valued on many committees, where his capacity for logical and tolerant debate made his opinions highly respected. He was honorary medical secretary of the South Australian Branch of the British Medical Association in 1927-1929, and President of the Branch in 1933-1934. In 1928 Britten Jones and F. N. Le Messurier were responsible for the origin and establishment of the Adelaide Post-Graduate Committee, and he continued to exert his influence until that organization was taken over by the Council of the University in 1943.

These activities made him widely known; but they may be forgotten in the passage of time. His enduring qualities were personal. In his youth a brilliant athlete and interstate footballer, he played a good game of cricket, tennis and golf until the second World War, with the keenest enjoyment not only to himself but also to his opponents; he was modest in victory, and in defeat the most generous loser. He loved good company, and one of his greatest pleasures was a holiday with his colleagues in a boat, a lot of amateur fishing and perhaps a little very skilful gambling; his judgement on a racecourse was as uncanny as his judgement in medicine. He had all the attributes of a really great man—great charm of manner, humility, modesty and kindness of heart, with a deeply religious foundation. Small wonder that this man, with so much humanity and kindness, was held in such respect and affection by the whole community, and was beloved of those who knew him intimately.

In 1915 he married Hilda, the younger daughter of the late Frank Fisher, and to her and her family we offer the deepest sympathy in their sudden bereavement. "Teddy B.J." and his wife were attending the Rhodes Scholars' reunion at Oxford in June when he had a minor cerebral vascular accident. But the end came with no warning; indeed, he had been visiting patients an hour beforehand. One can only be thankful that he was spared the suffering of a lengthy cerebral illness, and we can remember him while all his most endearing qualities were in evidence.

Dr. F. N. Le Messurier writes: As a personal friend of the late Sir Edmund Britten Jones I would like to add tribute to his many outstanding qualities. My first association with him was in the year 1908, when we played together as members of the North Adelaide Football Club. He always played the game in the spirit of sportsmanship that was so characteristic of his dealings on and off the playing fields. He was extremely popular among his fellow players, and his sense of humour, which was to be enjoyed so much by his professional friends later, was even noticeable at that stage of his career. He was also a good cricketer and bowled with considerable success for the Adelaide University Eleven; later in his life at Oxford he became quite a good bat. It was a lucky day for the Adelaide Children's Hospital when he was appointed a member of the honorary medical staff, as undoubtedly his pleasing temperament and disposition played a prominent part in making this hospital such a happy institution for his professional colleagues. It was recognized at an early date that he had one of the best balanced minds in his profession. Consequently his opinion was sought for not only in the wards, but also at the meetings of the honorary medical staff, when future policies of the hospital were decided. He was very considerate to his house surgeons and students and was always ready to help them with any problem, medical or social, that might confront them. He earned the esteem of the nursing staff as their medical adviser, and all those who worked in his wards considered themselves fortunate. He was thoughtful for their welfare and was understanding in their difficulties. His sudden passing has left a gap which will be difficult to fill.

Dr. Mark Bonnin writes: As a final tribute to the late Sir Edmund Britten Jones I do not wish to write a biography but rather to express the sentiments of many of the younger physicians of Adelaide. Our first contact with "Teddy", as he was affectionately known to his students as well as his colleagues, was in the wards of the Adelaide Children's



Hospital. There he was always regular in attendance, and the round started punctually. Even in those days as students we were impressed with his wisdom and sound judgement on clinical problems. The rounds were not too long in duration, and the teaching was brief and to the point, so that at the end of the round one felt that one had acquired lasting knowledge from a few points well made.

My next contact with Sir Edmund was in the Middle East, where he was O/C Medical Division of the 2/2 Australian General Hospital. There his extensive clinical experience was put to good use as a medical consultant. The medical division was run "without a hitch", a tribute to his administrative ability. I first learned to respect him for his encouragement to younger graduates when, as a general duties captain, I was entrusted with the responsibilities of seeing patients in consultation on his behalf. This may seem a trivial fact to mention, but to me at the time it was a tremendous stimulus and a compliment that will never be forgotten. It is mentioned merely as one example of the way in which he constantly encouraged and helped his juniors in their professional advancement.

After the war many returned graduates were uncertain as to their future livelihood. I naturally turned to Sir Edmund for advice, and from then until his untimely death I frequently sought his guidance on matters concerning my professional career. He always found time to give his advice, and this in spite of the fact that he was perhaps the busiest consultant that ever practised in South Australia. All graduates seeking advice were received with courtesy, and the resources of his able mind were made available to them. Indeed many returned men are quite unaware of the debt they owe to him. In his capacity as a member of the Post-Graduate Committee in Medicine he was an untiring worker on their behalf in the immediate post-war years. It was

largely through his offices, together with his friends on the committee, particularly Dr. Stanley Verco, Dr. Ray Hone and Mr. Ivan Jose, that the Reconstruction Training Scheme programme for doctors was rapidly and efficiently put into being.

During the period after the war when professional rooms were not readily available he was kind enough to allow me the use of one of his consulting rooms, where I worked for some two years and was in contact with him daily. During that period or at any other time I did not hear one disparaging remark about him from any of his patients, his colleagues or members of his staff. His honesty, fairness and courtesy in dealing with all made it virtually impossible for anyone to find fault, and to my knowledge he never made an enemy. I learnt to appreciate his true value as a consultant physician when he was asked to see a patient suffering from anuria with me. Not only had he the old world manner of inspiring confidence when dealing with patient and relatives alike, but it was his modern scientific knowledge that ultimately led to the recovery of the patient. Though few can hope to attain his eminence in professional and public life, it is to be hoped that every young physician will try to follow his example.

Dr. C. Yeatman writes: I am glad to add my sprig of rosemary (that's for remembrance) to the many tributes to Edmund Britten Jones. As students and residents at the hospital, I and all his associates recognized his exceptional mental powers, but it is of his disposition and temperament I wish to write. He had a joyous delight in a bit of fun, the subtle prank and leg-pull. More than that, much more, he possessed that lovely virtue "charity" as defined by St. Paul: "Charity suffereth long and is kind: charity envieth not: charity vaunteth not itself, is not puffed up, doth not behave itself unseemly, seeketh not its own, is not easily provoked."

I do not remember to have seen Teddy B.J. out of humour nor ever show any petty irritation. And now that his sojourn here is ended I recall the words of Francis Bacon: "But, above all, beleeve it, the sweetest canticle is, *Nunc Dimittis*, when a Man have obtained worthy Ends and Expectations."

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 79, of December 3, 1953.

NAVAL FORCES OF THE COMMONWEALTH

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Appointment.—Surgeon Commander John Howard Mitchell is appointed on loan from the Royal Navy, with seniority in rank of 30th June, 1952, dated 9th November, 1953.

Resignation. — The resignation of Gerald Vincent Mulholland of his appointment as Surgeon Lieutenant (for Short Service) is accepted, dated 31st October, 1953.

Honorary Physicians, Surgeons and Dental Surgeons.—Surgeon Commander (Acting) Robert Michael Coplans and Surgeon Commander Kenneth Charles Armstrong are appointed Honorary Physician and Honorary Surgeon, respectively, to His Excellency the Governor-General, for a period of three years, dated 14th October, 1953.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Confirmation in Rank.—Surgeon Lieutenants (on probation) Ian Douglas Wilson, Francis Victor Harder, Peter William Fay and Trevor Nelson Hatfield are confirmed in the rank of Surgeon Lieutenant, with seniority in rank of 21st May, 1948, 13th August, 1952, 1st September, 1952, and 1st September, 1952, respectively.

Appointment.—Kenneth Ivor Digwood is appointed Surgeon Lieutenant, dated 29th September, 1953.

Royal Australian Naval Volunteer Reserve.

Appointment. — Douglas Arthur Warden (Surgeon Lieutenant-Commander, Royal Australian Naval Reserve) is

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 5, 1953.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	6(4)	1(1)	..	..	..	..	..	..	7
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	1	..	1
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	1	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	2	7(7)	10(7)	..	..	..	..	..	19
Diphtheria	4(2)	..	3(2)	..	2(1)	..	..	..	9
Dysentery (Bacillary)	..	1(1)	..	..	..	..	..	..	1
Encephalitis	..	1	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	..	20(1)	..	..	6(5)	..	..	..	26
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	2(1)	2	..	..	2(2)	1	..	..	7
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	1(1)	..	..	..	..	1(1)	..	..	2
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	9(6)	2(1)	..	1(1)	..	..	..	..	12
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	5(2)	1(1)	..	38(21)	..	..	..	44
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	10(9)	25(17)	12(12)	2(1)	2(2)	..	..	1	52
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1	1	..	..	..	..	..	2
Trachoma	..	..	..	..	2	..	..	..	2
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	25(15)	17(8)	12(5)	11(9)	7(3)	2	..	..	74
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	1(1)	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

appointed Surgeon Lieutenant-Commander, with seniority in rank of 1st June, 1944, dated 30th September, 1953.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—1/39057 Captain H. H. Chesterfield-Evans is seconded for post-graduate studies in the United Kingdom, 1st December, 1952.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/152043 Captain J. Laing is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District), 19th August, 1953. 2/52118 Major E. H. Hipsley is absorbed in a vacancy within the authorized establishments of Majors, and to receive pay and allowances of that rank, 24th June, 1953.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/63671 Lieutenant-Colonel K. H. Heard relinquishes command of 4th Field Ambulance, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District), 28th August, 1953. 3/114023 Major (provisionally) C. A. Jones is seconded for post-graduate studies in the United Kingdom, 9th February, 1953.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

1st Military District.—To be Honorary Captains, 20th October, 1953: Peter Gray Mitchell and William James Hamilton.

2nd Military District.—To be Honorary Captain, 22nd October, 1953: Frank Samuel Lucas.

3rd Military District.—To be Honorary Captains, 11th September, 1953: Henry Alfred Jenkins and William Arthur Self.

5th Military District.—To be Honorary Captain, 12th September, 1953: Graham Raymond Raad.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

Squadron Leader L. A. Watson (034058) is transferred to the Reserve, 21st October, 1952, on completion of his short-service commission.

No. A15209 Air Cadet Colin Robert George Stoddart (015209) is appointed to a short-service commission on probation for a period of twelve months, 5th October, 1953, with the rank of Flight Lieutenant.

The resignation of Flight Lieutenant J. F. Marrington (021966) is accepted, 14th October, 1953.

Air Force Reserve.

Medical Branch.

The following former officers are appointed to commissions with the rank of Flight Lieutenant: A. C. Schwieger (257345), 22nd September, 1953; P. M. Birrell (297470), 22nd October, 1953.

The appointment of Squadron Leader R. D. G. Vann (024187) is terminated, 23rd October, 1953.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Course in Occupational Medicine.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a course in occupational medicine will be held for a period of one to two weeks beginning late May or early June, 1954. Those interested in attending are requested to communicate with the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483.

Deaths.

THE following deaths have been announced:

SABIEL.—Frederick Herman Sabiel, on December 19, 1953, at Sydney.

NORTH.—Robert Bell North, on December 23, 1953, at Sydney.

WHITEMAN.—Reginald John Whiteman, on December 25, 1953, at Lewisham, New South Wales.

O'HALLORAN.—Patrick Leo O'Halloran, on December 26, 1953, at Inverell, New South Wales.

Diary for the Month.

JAN. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JAN. 15.—Queensland Branch, B.M.A.: Council Meeting.
JAN. 18.—Victorian Branch, B.M.A.: Finance Subcommittee.
JAN. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.